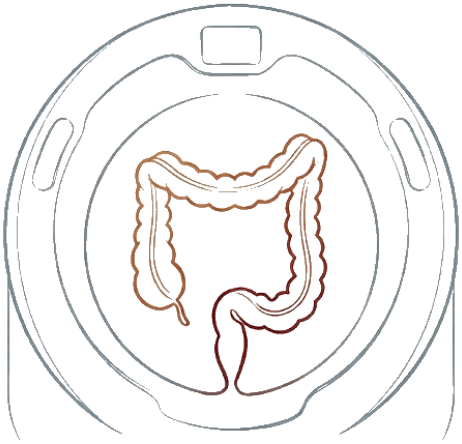
CT Imaging for Colorectal Surgery



Graham Bay, MD

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v2

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Audience: Colorectal Surgery Residents **Format:** Lecture handout – companion to case-based presentation

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Objectives

1. Technical Fundamentals – CT technique, contrast, image acquisition
2. Colorectal Carcinoma – Staging, pitfalls, key lessons
3. CT Colonography – Protocol, limitations, pitfalls
4. GI Bleed – Active bleed detection, CTA technique
5. Ischemic Gut – Types, key signs, stercoral colitis
6. Inflammatory Bowel Disease – UC vs Crohn's, extra-GI findings
7. Pitfalls, Mimics & Miscellaneous – Mucocele, internal hernia, epiploic appendagitis, omental infarct
8. CT Fluoroscopy Procedures

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1 Technical Fundamentals

1.1 Contrast – Why It Matters

1.1.1 IV Contrast Phases

The single most important technical concept for surgeons to understand: **which phase you scan determines what you can see.**

Portal Venous Phase (60-70 sec) – The Workhorse

- Standard phase for virtually all colorectal imaging.
- Contrast has passed through arterial system, through capillary beds, and is now draining into the portal venous system and hepatic veins.
- **Why it matters for colorectal surgery:**
  - Optimal liver parenchyma enhancement – best single phase for detecting colorectal liver mets (typically hypovascular, appearing as low-density lesions against brightly enhancing liver).
  - Good opacification of mesenteric veins (SMV, IMV) – critical for surgical planning.
  - Adequate bowel wall enhancement – allows assessment of wall thickening, inflammation, tumor enhancement.

> **TIP:** "If you only get one phase for staging colorectal cancer, this is it."

Arterial Phase (25-30 sec)

- Contrast is in the arteries but has not yet reached the portal venous system.
- **When you need it in colorectal practice:**

  - **Active GI bleeding (CTA):** extravasation requires arterial phase. Detects bleeding as slow as 0.3-0.5 mL/min.
  - **Hypervascular liver mets:** some colorectal mets (particularly neuroendocrine tumors of the colon/rectum) are hypervascular and best seen on arterial phase.
  - **Vascular mapping:** SMA, IMA, and their branches before complex resections. Identifying a replaced right hepatic artery off the SMA (present in ~15-20% of patients – critical for hepatic resection planning).
  - **Mesenteric ischemia workup.**

Delayed Phase (3-15 min)

- Contrast has equilibrated and is being excreted by the kidneys. Ureters and bladder are opacified.
- **When you need it:**
  - **Ureteral involvement:** locally advanced rectal or sigmoid tumors can encase/obstruct ureters.
  - **Bladder invasion:** T4b rectal tumors invading bladder. Delayed phase shows contrast-filled bladder lumen and tumor extension.
  - **Slow venous bleeding:** sometimes catches extravasation not seen on arterial phase.

> **TIP:** "If you are worried about ureteral proximity or bladder invasion, specifically request delayed images – they are NOT part of a standard CT."

Quick Reference Table:

Phase	Timing	Key Use in Colorectal
Arterial	25-30 sec	GI bleed, vascular mapping, hypervascular mets
Portal Venous	60-70 sec	Standard staging, liver mets, mesenteric veins
Delayed	3-15 min	Ureteral/bladder involvement, slow bleed

1.1.2 Types of IV Contrast

- **Non-ionic, low-osmolality agents** (iohexol/Omnipaque, iopamidol/Isovue) are the current standard. Reaction rate ~1-3% mild.
- Standard dose: ~100-150 mL for an average adult at 2-4 mL/sec.
- **Contrast reactions** are "allergic-like" (ACR terminology; not true IgE-mediated) – prior uneventful exposure does NOT protect. Prior reaction = 5-6x increased risk; premedicate with prednisone 50 mg PO at 13h, 7h, 1h before + diphenhydramine 50 mg.
- **Contrast nephropathy (PC-AKI):** Risk is much lower than historically believed for IV contrast. Current guidelines: IV contrast generally safe with eGFR >30. For eGFR <30, discuss risk/benefit and hydrate.

> **TIP:** "Do not let fear of contrast nephropathy delay a critically needed contrast CT. The risk of missing the diagnosis almost always outweighs the renal risk. Hydrate and scan."

1.1.3 Oral Contrast

Positive oral contrast (appears white):

- **Barium sulfate:** traditional positive oral agent; still used in some centers for bowel opacification. Many institutions now omit positive oral contrast for staging CTs, relying on IV contrast and neutral oral agents instead. Contraindicated if perforation suspected (causes chemical peritonitis).
- **Gastrografin (water-soluble iodinated):** use when perforation suspected. Also has a therapeutic role in adhesive SBO – hyperosmolar, draws fluid into lumen, can reduce need for surgery and shorten hospital stay.

> **PEARL FOR SURGEONS:** "The Gastrografin challenge for SBO is both diagnostic AND therapeutic – if contrast reaches the cecum within 8-24 hours, the obstruction is likely to resolve nonoperatively."

Neutral oral contrast (water/VoLumen):

- Distends bowel without opacifying the lumen. Better visualization of the enhancing bowel wall.
- Increasingly preferred for CRC staging to better assess bowel wall enhancement and tumor conspicuity.

1.1.4 Rectal Contrast

- Water-soluble rectal contrast for **suspected anastomotic leak** (extravasation = leak).
- CO2 or air for CT colonography.
- Rectal contrast to demonstrate colovesical or colovaginal fistulae.

> **TIP:** "When ordering a CT for possible anastomotic leak, specifically request rectal water-soluble contrast – it will NOT be done unless asked for."

1.2 Image Acquisition – Helical vs Axial

Axial (Step-and-Shoot) – the original method:

- Tube rotates 360 degrees, acquires one slice, table moves, repeat.
- Limitations: gaps between slices, longer scan times, respiratory misregistration, poor reformats.

Helical (Spiral) – the current standard:

- Tube rotates continuously while table moves continuously, tracing a helical path.
- Entire abdomen/pelvis in a single 5-10 second breath-hold.
- **Volumetric data** = no gaps. This enables high-quality multiplanar reformats (coronal, sagittal) from a single acquisition with no additional radiation.

Key Parameters:

- **Pitch:** table travel per rotation / beam width. Pitch ~1 = standard. Higher = faster but noisier. Lower = better quality but more dose.

- **Overlapping reconstruction:** reconstructing slices at intervals smaller than slice thickness (e.g., 5 mm slices every 2.5 mm). Does NOT increase radiation. Improves reformat quality and reduces partial volume artifact.
- **Typical reconstruction for CRC staging:** thin axial (1.25-3 mm) with coronal and sagittal reformats. Thick (5 mm) also generated for primary review.

1.3 Image Planes – Why Coronal Reformats Are Essential

Axial (transverse): the traditional plane. Best for cross-sectional anatomy, measuring wall thickening, relationship to adjacent structures.

Coronal: arguably the most important reformat for colorectal surgery planning.

Why:

1. **Following mesenteric vessels** – SMA, SMV, IMA, IMV all run predominantly craniocaudal and are beautifully displayed on coronal. You can trace the IMA from its aortic origin (L3, ~3-4 cm above bifurcation) through left colic, sigmoid branches, and superior rectal artery.
2. **IMV landmark** – runs to the left of the IMA, drains into splenic vein. At risk during medial-to-lateral mobilization of left colon. Coronal shows entire course.
3. **Variant anatomy** – replaced right hepatic artery off SMA (~15-20%) easily seen on coronal. Critical for liver resection planning.
4. **Mesenteric lymph nodes** – chains along IMA and SMA seen in their native plane. Easier to count, assess size, and determine relationship to named vessels.
5. **Bowel obstruction transition points** – dilated proximal and collapsed distal bowel often visible on the SAME coronal image. Much easier than following loops on axial.
6. **Peritoneal carcinomatosis** – omental caking, paracolic gutter deposits, pelvic implants give a gestalt of disease burden helpful for PCI estimation.

> **TIP:** "When you call about an obstruction, I look at the coronals first for the transition point, then confirm on axials."

Sagittal: indispensable for rectal cancer.

- Shows tumor relationship to sacrum, mesorectal fascia, bladder/prostate/uterus anteriorly.
- "Surgical view" for LAR or APR planning.
- Best plane for presacral masses and sacral invasion.

1.4 Scout Image (Topogram)

A low-dose, single-projection radiograph obtained at the start of every CT. Takes 2 seconds to review.

Always review it because:

1. **SBO:** dilated loops and air-fluid levels immediately apparent.
2. **Free air:** lucency over liver or outlining bowel loops/falciform ligament.
3. **Lung bases:** pleural effusions, consolidation, lung nodules, rib lesions. Pulmonary mets are second most common distant site after liver.
4. **Bone lesions:** lytic/blastic mets, pathologic fractures, sacral destruction from locally advanced rectal cancer.
5. **Lines and tubes:** NG tubes, ureteral stents, drains, ports – instant overview.
6. **Scan planning verification:** ensures coverage from diaphragm through perineum for CRC staging.

> **TIP:** "The scout takes 2 seconds to review and can immediately reveal SBO, free air, or bone mets. Always start with the scout."

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2 Colorectal Carcinoma

2.1 Staging on CT – What You Need to Know

2.1.1 T Staging

- **T1 and T2 are indistinguishable on CT.** Both appear as wall thickening confined to the bowel wall. Do not attempt to differentiate – this is a pathologic distinction. For early local staging (T1 vs T2), endorectal ultrasound (EUS) is preferred over MRI. MRI's strength begins at the T2/T3 boundary (integrity of the muscularis propria) and CRM assessment.
- **T3:** tumor extends through muscularis propria into pericolonic fat. Look for **irregular soft-tissue stranding or nodularity** in the fat immediately adjacent to the tumor.
- **T4a:** visceral peritoneal involvement. CT clues: focal peritoneal thickening, nodularity on serosal surface.
- **T4b:** invasion of adjacent organs. **Loss of fat plane** between tumor and bladder, uterus, prostate, small bowel, or abdominal wall.

> **TIP:** "If I can see fat between the tumor and your structure of interest, you are likely safe. If I cannot, prepare for a more complex operation."

2.1.2 N Staging

- Short axis >1 cm is the traditional threshold, but has poor sensitivity (~50%) and specificity (~70%).
- **Morphology matters more than size:**
  - Round shape (normal nodes are oval/reniform)
  - Heterogeneous enhancement
  - Irregular or indistinct borders
  - Central necrosis

> **PEARL:** "A 6mm round node with an irregular border worries me more than a 12mm bean-shaped node with a fatty hilum."

- **Where to look:** follow the mesenteric vessels on coronal reformats. Nodes cluster along ileocolic, right colic, middle colic, left colic, and SMA/IMA.
- Overall accuracy for N staging: only ~60-70%. CT cannot detect microscopic metastases.

2.1.3 M Staging

- **Liver mets:** typically hypovascular, appearing as hypodense lesions on portal venous phase. Peripheral rim enhancement on arterial phase (desmoplastic reaction). Report number, size, Couinaud segment, relationship to hepatic veins and portal pedicles.
- **Lung mets:** multiple round, well-defined peripheral nodules of varying sizes.
- **Peritoneal carcinomatosis:** omental caking, ascites, peritoneal nodularity, liver/spleen surface scalloping. Stage IVC. Changes surgical planning dramatically.

2.1.4 Key Surgical Reporting for Rectal Cancer

Always include:

1. **Distance from anal verge:** Low (0-5 cm), Mid (5-10 cm), Upper (10-15 cm) – determines surgical approach.
2. **Relationship to anterior peritoneal reflection:** above vs below changes CRM considerations and treatment decisions.
3. **Invasion of adjacent structures:** bladder, uterus/vagina, prostate/seminal vesicles, pelvic sidewall, sacrum.
4. **Circumferential extent:** clock-face position.

> **TIP:** "Surgeons need three things from the radiologist on a rectal cancer: how far from the anal verge, above or below the peritoneal reflection, and what structures are at risk."

2.1.5 Role of MRI in Rectal Cancer

MRI is the **gold standard** for local staging. CT stages the body; MRI stages the rectum.

- **CRM / Mesorectal Fascia (MRF):** the single most important predictor of local recurrence. Threatened CRM = tumor or node within 1 mm of MRF. Drives the neoadjuvant chemoradiation decision.
- **EMVI (Extramural Vascular Invasion):** tumor signal replacing normal dark flow void in mesorectal vessels. Independent predictor of distant mets and poor survival.
- **DWI (Diffusion-Weighted Imaging):** now standard in rectal cancer MRI protocols. Highlights cellular tumors as bright signal against suppressed background. Critical for **post-neoadjuvant response assessment** – persistent restricted diffusion at the tumor bed suggests residual viable tumor, while signal normalization suggests complete pathologic response (pCR).
- **Low rectal cancers:** MRI assesses relationship to intersphincteric plane, external sphincter, levator ani – critical for sphincter-preservation decisions.

> **TIP:** "Every rectal cancer deserves an MRI before treatment decisions are finalized. The CRM is the number one thing I look for – it drives the neoadjuvant decision."

2.1.6 Apple-Core Lesion

- Classic annular colorectal carcinoma: **short segment (3-6 cm) of circumferential wall thickening** with irregular luminal narrowing, shouldered/overhanging edges, and mucosal ulceration.
- DDx: diverticular stricture (longer, smoother), Crohn's stricture (mural stratification), lymphoma (may be aneurysmal), ischemic stricture (watershed).

> **PEARL:** "Short, irregular stricture with shouldered edges = cancer until proven otherwise."

2.2 Case-Based Teaching Points

2.2.1 Subtle Colon Cancers

Not all colon cancers are obvious apple-core lesions. Some present as subtle focal wall thickening with minimal luminal narrowing. **Key lesson:** Follow the mesenteric vessels on coronal reformats to find associated lymphadenopathy that may be the first clue to an otherwise subtle primary.

2.2.2 Identifying the Correct Primary Site

Getting the primary site right matters enormously – it drives the entire treatment pathway. A **sigmoid cancer invading bladder** is treated with surgery first, whereas a **bladder cancer invading sigmoid** is treated with chemoradiation first. Misidentifying the primary can delay appropriate treatment by weeks.

2.2.3 Mesenteric Nodes on Coronal Reformats

Mesenteric lymphadenopathy is easily missed on axial images alone. Coronal reformats display mesenteric node chains in their native plane along the IMA and SMA, making them far more conspicuous. **Always scroll through coronals looking for nodes.**

2.2.4 Obstructing Carcinoma

Always correlate CT with plain films when available. The plain film may show the obstruction pattern (dilated loops, air-fluid levels) that provides context for the CT transition point.

2.2.5 Anastomotic Recurrence

Post-operative recurrence at the anastomotic site is one of the most commonly missed findings on CT. **Always scrutinize the anastomosis on follow-up imaging.** On MRI, look for intermediate T2 signal soft tissue that restricts on diffusion-weighted imaging.

2.2.6 Calcified Mesorectal Nodes

Calcified nodes in the mesorectum are unusual for reactive nodes. In the setting of a rectal primary, calcified nodes can represent **mucinous metastases** (mucinous adenocarcinoma can produce calcification). Do not dismiss calcified nodes as benign in this context.

2.2.7 Non-Adenocarcinoma Colonic Masses

Not every colonic mass is adenocarcinoma. Large retroperitoneal or mesenteric masses with fat and soft-tissue components suggest **liposarcoma**. Dedifferentiated liposarcoma has aggressive non-lipomatous components that may invade adjacent organs including colon.

2.2.8 Polyposis Syndromes in Young Patients

Polyposis syndromes (FAP, attenuated FAP, MUTYH-associated) presenting in young patients warrant full CT staging including assessment for desmoid tumors (mesenteric), duodenal adenomas, and thyroid nodules.

2.2.9 Lung Base Review on Chest CT

The lung bases are included on chest CT. Colon masses in the transverse colon and hepatic/splenic flexures are visible on chest CTs but frequently overlooked because the radiologist is focused on the lungs. **Always glance at the upper abdomen on chest CTs.**

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3 CT Colonography

3.1 How It Works

Patient Preparation:

- Low-residue diet 1-3 days prior.
- Cathartic bowel prep (same goal as optical colonoscopy – a clean colon).
- **Fecal and fluid tagging:** patient ingests oral barium sulfate and/or iodinated contrast with meals 1-2 days before. This labels residual stool/fluid as bright white, distinguishing it from true soft-tissue polyps.

The Scan:

- Small rectal catheter placed. **Automated CO2 insufflator** (e.g., PROTOCO2L) distends the colon. CO2 is used rather than room air because it is rapidly absorbed across colonic mucosa – significantly less post-procedure cramping.
- **Dual-position acquisition:** supine AND prone. Critical for redistributing gas to maximize segment visualization.

- **Low-dose technique:** ~3-6 mSv total (both acquisitions) vs 10-20+ mSv for standard contrast-enhanced CT.
- **No IV contrast.**

Parameter	Typical Value
Slice thickness	0.5-1.25 mm
kVp	120 (100 in smaller patients)
mAs	50 or less (low dose)
IV contrast	None
Positions	Supine + Prone
Insufflation	Automated CO2

3.2 Why CTC is NOT a Replacement for Diagnostic CT

This is critical for surgeons to understand.

CTC is optimized for one thing: **detecting polyps and masses on the colonic surface.** It sacrifices everything else for that.

- **No IV contrast** = cannot characterize solid organ lesions, assess vasculature, or evaluate enhancement patterns.
- **Low-dose technique** = noisy images, insufficient for diagnostic-quality soft tissue evaluation.
- **Cannot stage a known cancer:** cannot assess T-stage, N-stage, or M-stage.

> **TIP:** "When CTC finds a large mass, do NOT skip the staging CT. The CTC told you something is there; the staging CT tells you what to do about it."

3.3 Distension – The #1 Technical Factor

Inadequate distension is the single most common cause of a non-diagnostic CTC.

- A collapsed segment hides polyps and can itself mimic a mass or stricture.
- **Most prone to collapse:** sigmoid (redundancy, muscular thickening, dependent position), transverse (mobile mesentery), rectum.
- **How supine/prone helps:** gas rises to non-dependent segments. Combining positions maximizes the chance every segment is adequately distended on at least one series.

> **TIP:** "If the report says a segment was 'not adequately distended on either acquisition,' that segment has NOT been evaluated. That is a blind spot that may need endoscopic assessment."

3.4 Tagging

- Oral barium/iodine labels residual stool/fluid as bright white (>200 HU).
- True polyps are soft tissue density (~30-60 HU) and will NOT tag.
- **Good tagging:** >90% residual material uniformly white, confident polyp detection.
- **Bad tagging:** intermediate-density stool, multiple equivocal findings, reduced specificity.

3.5 Key Limitations and Pitfalls

- **Flat lesions (sessile serrated):** the Achilles' heel. Only slightly elevated above mucosa. CTC sensitivity drops significantly. Important because these are precursors to right-sided cancers via the serrated neoplasia pathway.
- **Lesions behind folds:** haustral folds create blind spots. 3D fly-through in both directions helps.
- **Diverticular disease:** thickened folds, inverted diverticula, trapped stool can all mimic polyps. Severe diverticular disease makes sigmoid essentially non-evaluable by CTC.
- **Extracolonic findings:** CTC can detect large findings (aortic aneurysm, large renal masses) but CANNOT reliably assess small liver lesions, peritoneal disease, or subtle adrenal/renal lesions. C-RADS E classification (E1-E4) used for reporting.

> **PEARL FOR SURGEONS:** "If your patient has severe sigmoid diverticular disease, CTC will have limited sensitivity in that segment. But CTC may be valuable for evaluating the proximal colon if the scope cannot traverse a narrowed sigmoid."

C-RADS Quick Reference:

- C1: No polyps or <6 mm – continue screening
- C2: 6-9 mm – surveillance CTC in 3 years or colonoscopy
- C3: >=10 mm or >=3 polyps 6-9 mm – colonoscopy with polypectomy
- C4: Colonic mass, likely malignant – surgical consultation

3.6 Case-Based Teaching Points

3.6.1 CTC Failure Due to Stricture

CTC is often ordered to evaluate proximal colon after incomplete colonoscopy due to obstruction. However, the stricture itself limits distension proximally, rendering upstream segments unevaluable. **If CTC proximal to an obstructing lesion is non-diagnostic, consider intraoperative palpation or early post-op colonoscopy (3-6 months) to clear the proximal colon.**

3.6.2 Ischemic Stricture vs Malignant Stricture

Ischemic strictures are typically smoother, longer, and in watershed distribution. Malignant strictures are shorter with shouldered edges. Overlap exists – when in doubt, scope it.

3.6.3 Diverticulitis vs Perforated Sigmoid Cancer

One of the most important differential diagnoses in abdominal imaging. Both present with sigmoid wall thickening, pericolonic stranding, and possible perforation.

Clues favoring cancer over diverticulitis:

- Mass effect or shouldered edges
- Regional lymphadenopathy
- Liver metastases
- Longer-segment asymmetric wall thickening

Key statistic: 2-11% of patients with complicated diverticulitis have CRC found on follow-up colonoscopy. **Follow-up colonoscopy after resolution of acute inflammation (typically 6-8 weeks) is essential to exclude cancer.**

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4 GI Bleed

4.1 CTA Protocol for GI Bleed

Three-Phase Protocol:

Phase	Timing	Purpose
Non-contrast	Before injection	Baseline: identify pre-existing hyperdensity (calcifications, retained contrast, clips)
Arterial	25-35 sec	Detect active arterial extravasation
Portal venous	60-70 sec	Detect venous bleeding, confirm arterial bleed that pools
Delayed (optional)	3-5 min	Slow/intermittent bleed; contrast pooling

What active extravasation looks like:

- Hyperdense focus (matching arterial density, >90 HU) within bowel lumen on arterial phase that was **NOT present on non-contrast.**
- On portal venous/delayed: contrast **increases in volume and changes shape** (pools, layers dependently, tracks along lumen).
- “Jet sign”: linear high-density streak from vessel into lumen.

> **PEARL:** "Active bleed = new hyperdensity on arterial phase that gets bigger on delayed phase. If it was there on non-contrast, it is not bleeding – it is a mimic."

4.2 Distinguishing Bleed from Mimics

	Active Bleed	Mimic
Present on non-contrast?	NO	YES
Changes between phases?	YES (grows, pools)	NO
Density	Matches aorta	Calcification: very high HU; clips: metallic

Common mimics: calcified phleboliths, retained oral contrast/barium, surgical clips/staple lines, hyperdense stool.

> **PEARL:** "The non-contrast phase is your best friend for ruling out mimics."

4.3 Sensitivity and Limitations

- CTA detects bleeding rates as low as **0.3-0.5 mL/min** (modern scanners possibly 0.1 mL/min).
- Overall: **sensitivity 85-90%, specificity 92%.**
- **Key limitation:** requires active bleeding at the time of scanning. A negative CTA does not exclude a bleeding source.

4.3.1 Nuclear Medicine: Tagged RBC Scintigraphy

When CTA is negative but clinical suspicion remains high, **Tc-99m tagged RBC scintigraphy** is the next step.

- **Detection threshold:** 0.1-0.4 mL/min – more sensitive than CTA for slow or intermittent bleeding.
- **Strength:** can image over hours (delayed images up to 24h), capturing intermittent bleeds that a single CTA snapshot misses.
- **Weakness:** poor anatomic precision – localizes to a general region (e.g., "right lower quadrant activity") but cannot name a specific artery or segment reliably. Intraluminal blood can transit, leading to false localization.

- **Role:** best used as a bridge – confirms active bleeding and guides timing for angiography or repeat CTA.

> **TIP:** "CTA is the first-line test for acute GI bleed because it localizes precisely. If CTA is negative, tagged RBCs can catch the intermittent bleeder – but tell the surgeon the localization is approximate."

4.4 Causes of Lower GI Bleed

Cause	Frequency	CTA Clues
Diverticular	~40%	Extravasation into diverticulum. Usually right-sided (ascending diverticula bleed more).
Angiodysplasia/AVM	~25%	Hypervascular nodular vessels in wall; early draining vein. Cecum/ascending. Older patients.
Post-surgical anastomotic	Variable	Bleed at staple/suture line. History key.
Neoplasm	~12.5%	Mass with extravasation.
Ischemic	Variable	Segmental wall thickening in watershed distribution.

> **PEARL:** "Right-sided diverticula bleed; left-sided diverticula get infected."

4.5 Reporting a CTA GI Bleed

1. **Is there active extravasation?** Yes/No. Phase first appears and how it changes.
2. **Location:** specific bowel segment using anatomic landmarks.
3. **Arterial supply – NAME THE ARTERY.** Use MIP reconstructions to trace:
    - Ileocolic artery (cecum, terminal ileum)
    - Right colic artery (ascending)
    - Middle colic artery (transverse)
    - Left colic / marginal artery of Drummond (descending)
    - Sigmoid branches of IMA
    - Superior rectal artery (upper rectum)
4. **Active vs recent:** active = contrast extravasation; recent = "sentinel clot" (high-density intraluminal blood 60-80 HU without frank extravasation).
5. **Underlying cause if visible:** mass, diverticulum, AVM.

> **TIP:** "Name the artery. If IR is going to embolize, they need to know which branch to select."

4.6 Case-Based Teaching Points

4.6.1 CTA Detecting Bleeds Missed at Colonoscopy

CTA can detect active bleeding that colonoscopy misses, particularly when blood obscures the endoscopic field. **The delayed phase may be the key acquisition** – some bleeds are only apparent as pooling contrast on delayed images. Naming the arterial supply on CTA directly guides the surgical decision (e.g., right hemicolectomy for ileocolic artery territory bleed).

4.6.2 Anastomotic Bleeding

Post-surgical anastomotic bleeding can be subtle on CTA. The **sentinel clot sign** (high-density intraluminal material adjacent to the anastomosis without frank extravasation) may be the only finding if active bleeding is intermittent.

4.6.3 Hypervascular Bowel Wall Lesions

Hypervascular lesions in the bowel wall on arterial phase have a differential diagnosis that includes AVM/angiodysplasia, GIST (typically enhance briskly), and neuroendocrine tumors (characteristically hypervascular). Clinical context and morphology help distinguish.

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5 Ischemic Gut

5.1 Types of Mesenteric Ischemia

5.1.1 Arterial Occlusive (SMA Embolus/Thrombosis)

- **SMA embolus:** most common cause of acute mesenteric ischemia. Typically lodges just distal to the origin of the middle colic artery (~3-8 cm from SMA origin). Cardiac source (A-fib, recent MI, valvular disease) in majority.
- **SMA thrombosis:** occurs at vessel origin, superimposed on chronic atherosclerotic disease. History of “intestinal angina” (postprandial pain, food fear, weight loss).
- **CT hallmark: paper-thin bowel wall.** No blood getting in = no edema or thickening. Non-enhancing wall.
- Late signs: pneumatosis, portal venous gas = transmural necrosis.

> **TIP:** “A paper-thin, non-enhancing bowel wall is arterial ischemia. This bowel is dead or dying – it has no blood supply to even swell up.”

5.1.2 Non-Occlusive Mesenteric Ischemia (NOMI)

- ICU patients, low-flow states, vasopressors causing splanchnic vasoconstriction.
- **Watershed distribution:** splenic flexure and rectosigmoid junction.
- **Vessels are PATENT** – this is the key distinction. Diagnosis = clinical context + watershed distribution + patent vessels.

> **TIP:** "Intubated patient on three pressors, distending belly, ischemic splenic flexure with patent vessels – that is NOMI."

5.1.3 Venous Thrombosis (SMV/Portal Vein)

- More insidious, days of vague pain. Hypercoagulable states, cirrhosis, pancreatitis, malignancy.
- **Fundamentally different from arterial:** the bowel wall is THICK (blood gets in but cannot get out). Target sign with submucosal edema/hemorrhage. Mesentery looks "wet" with marked edema.
- **Look for the clot:** filling defect in SMV or portal vein on portal venous phase. Vein may be expanded.

> **TIP:** "Thick wall + mesenteric haziness + clot in the SMV = venous ischemia. Many of these patients can be managed with anticoagulation rather than immediate surgery, IF there is no frank necrosis or perforation."

Quick Distinction Table:

Feature	Arterial Occlusive	NOMI	Venous Thrombosis
Wall thickness	Paper-thin	Variable	Thick (edematous)
Wall enhancement	Absent/poor	Patchy/poor	Often preserved
Mesentery	Haziness (late)	Variable	Marked edema
Vessels	SMA thrombus/cutoff	Patent	SMV/PV thrombus
Distribution	SMA territory	Watershed	SMV drainage territory
Onset	Acute, sudden	Insidious (ICU)	Subacute (days)

5.2 Key CT Signs of Ischemia

- **Thin wall = arterial. Thick wall = venous.** The single most important distinction.
- **Pneumatosis intestinalis:** gas within bowel wall. Ominous in ischemia context. But benign pneumatosis exists (post-procedure, steroids, COPD). Pneumatosis + clinical concern = surgical emergency.
- **Portal venous gas:** branching lucencies extending to liver periphery (vs pneumobilia which is central). Late and ominous in ischemia.
- **Lack of enhancement:** non-enhancing wall = no blood supply = necrotic. Most reliable sign of irreversible ischemia.

- **Free fluid:** non-specific but supportive. Hemorrhagic ascites (high density) suggests advanced ischemia.

> **TIP:** "Always look at the vessels. On every CT where ischemia is a concern, scroll through the SMA and SMV on axial and coronal."

5.3 Malignant vs Adhesive (Bland) Obstruction

This is a critical distinction that changes surgical management.

Adhesive obstruction:

- Abrupt caliber change from dilated to decompressed bowel. No mass at transition point. Clean mesentery. History of prior surgery.
- "Beak sign" where bowel tapers at transition.
- Trial of conservative management appropriate (NGT, NPO, time).

Malignant obstruction:

- **Mass at the transition point.** Gradual transition over a segment. Mesenteric nodularity/carcinomatosis signs: omental caking, peritoneal nodules, ascites, lymphadenopathy. Multiple transition points suggest carcinomatosis.
- Conservative management unlikely to resolve. Plan for resection, bypass, or diversion.

> **TIP:** "If I see a mass at the transition point or peritoneal nodularity, I will call you and say this looks malignant – plan accordingly. If the transition point is clean and the mesentery is normal, it is likely adhesive and you have time to try conservative management."

Closed-loop obstruction (relevant to both): C-shaped dilated loop with two adjacent collapsed segments converging at a single point, mesenteric swirl/whirl sign, compromised wall enhancement. **Surgical emergency regardless of etiology.**

5.4 Stercoral Colitis

Pressure necrosis of the colonic wall from impacted, hard stool. Most common in rectosigmoid. Elderly, debilitated, opioid-using patients.

CT Findings:

1. Fecal impaction (prerequisite)
2. Colonic wall thickening adjacent to impacted stool
3. Pericolonic fat stranding
4. Focal wall defect if perforated
5. Extraluminal gas or fluid if perforated

Why it matters: often missed or dismissed as "just constipation." Stercoral perforation carries **30-50% mortality** (some series report up to 71%, driven by sepsis and patient comorbidities). Perforation typically at antimesenteric border of rectosigmoid.

By **Laplace's law** (wall tension = pressure x radius / wall thickness), the segment with the greatest fecal distension and thinnest wall is most vulnerable to perforation.

> **TIP:** "When you see massive fecal impaction with sigmoid wall thickening and pericolonic stranding, do NOT just write 'constipation.' That is stercoral colitis with real perforation risk. These patients need urgent disimpaction."

5.5 The Cecum in ICU Patients

Ogilvie's Syndrome (Acute Colonic Pseudo-Obstruction):

- Massive colonic dilatation without mechanical obstruction. Cecum and right colon most affected.
- ICU patients, post-surgery (especially orthopedic, cardiac), electrolyte abnormalities, opioids.
- Rectum often has gas (helps distinguish from mechanical obstruction).

Cecum is most vulnerable (thinnest wall, widest diameter). By **Laplace's law** (wall tension = pressure x radius / wall thickness), the cecum is where Ogilvie's perforates.

- **Graded risk by diameter:**
  - **>9 cm:** perforation risk begins to increase; warrants close monitoring.
  - **>12 cm:** high-risk; generally accepted threshold for mandatory intervention.
- **Duration matters:** sustained dilatation >9 cm for >3-6 days significantly increases risk even below 12 cm.
- **If cecal wall shows pneumatosis or becomes paper-thin:** perforation is imminent.

> **TIP:** "Every morning in the ICU, check the cecum. If it is over 12 cm, that is your problem. If it has been over 12 cm for days and is now showing wall thickening or pneumatosis, you need to act."

Management: Neostigmine (first-line) –> colonoscopic decompression –> surgery (cecostomy or right hemi) if ischemic/perforated.

5.6 Venous Ischemia – Pelvic Context

- Pelvic venous thrombosis (internal iliac vein, ovarian vein) can cause venous congestion of rectum, vagina, pelvic organs.
- **Rectal wall thickening with submucosal edema** in the setting of pelvic DVT can mimic proctitis.
- Post-surgical context: after pelvic surgery (APR, LAR, hysterectomy), pelvic vein thrombosis causes congestion of remaining structures.

> **TIP:** "If you see a thick-walled rectum with submucosal edema on the post-op CT, look at the pelvic veins. If there is a pelvic DVT, that thickening may be venous congestion – treatment is anticoagulation, not antibiotics."

5.7 Portal Hypertension as a Mimic

Portal hypertension causes diffuse bowel wall thickening that mimics ischemia/colitis.

How to distinguish from true ischemia:

- **Distribution:** portal hypertensive changes are diffuse and symmetric. True ischemia is segmental.
- **Associated findings:** cirrhotic liver, splenomegaly, varices, ascites.
- **Wall enhancement is preserved** (there is no ischemia). In true ischemia, enhancement is diminished.

> **TIP:** "Cirrhotic patient with entire small bowel wall thickened plus ascites and varices everywhere – that is portal hypertensive enteropathy, not ischemia. Do NOT take that patient to the OR. But if a cirrhotic patient has segmental wall thickening with mesenteric stranding AND a thrombus in the SMV – now you have both, and that IS a different story."

5.8 Case-Based Teaching Points

5.8.1 Venous Ischemic Enteritis

Distinguishing malignant vs adhesive obstruction changes management. Recognizing the **venous ischemia pattern** (thick wall, mesenteric edema, look for clot) is critical – it guides anticoagulation therapy rather than surgical exploration.

5.8.2 Ischemic Colitis in Young Patients

Ischemic colitis can occur in young patients and may not present in classic watershed distribution. Consider hypercoagulable states, vasculitis, and medication-related causes (oral contraceptives, cocaine) in younger demographics.

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6 Inflammatory Bowel Disease

6.1 UC vs Crohn's – The Key Distinctions

Feature	UC	Crohn's
Distribution	Continuous from rectum	Skip lesions, any GI segment
Depth	Mucosal/submucosal	Transmural
Ileal involvement	Backwash ileitis only (short)	Terminal ileum (classic)
Creeping fat	No	Yes (pathognomonic)
Fistulae/abscesses	No (except toxic megacolon perf)	Yes
Strictures	Rare (lead pipe narrowing)	Common
Comb sign	No	Yes
PSC association	Strong (10%)	Less common (5%)

Ulcerative Colitis Key CT Findings:

- Continuous, mucosal, always starts at rectum, extends proximally without interruption.
- **Lead pipe colon:** loss of haustral folds. Smooth, featureless, foreshortened tube.
- **Pseudopolyps:** islands of regenerating mucosa between ulcerated segments.
- **Backwash ileitis:** mild change in terminal ileum with pancolitis – short segment only. Long-segment ileal disease = think Crohn's.
- **Toxic megacolon:** transverse colon >6 cm with loss of haustration, thin wall, systemic toxicity. Look for pneumatosis, portal venous gas, free air. **Surgical emergency.**

> **PEARL:** "UC is a continuous, mucosal disease that always involves the rectum. If the rectum is spared or you see skip lesions, rethink the diagnosis."

Crohn's Disease Key CT Findings:

- **Transmural inflammation** causing the full spectrum: fistulae, abscesses, strictures, sinus tracts. On CT: mural stratification (target sign) with mucosal hyperenhancement, submucosal edema, serosal enhancement.
- **Skip lesions** – diseased segments separated by normal bowel. Pathognomonic.
- **Terminal ileum predominance** – the #1 site.
- **Fistulae:** enteroenteric, enterovesical (air in bladder is a clue), enterocutaneous, rectovaginal, perianal. Perianal fistulae best mapped with pelvic MRI.
- **Abscesses:** rim-enhancing collections. Report size, location, accessibility for percutaneous drainage.
- **Strictures:** the critical question is **inflammatory (enhancing, edematous) vs fibrotic (non-enhancing, thin wall)?** This determines medical vs surgical management. MR enterography with delayed contrast is best for this distinction.
- **Comb sign:** engorged vasa recta along mesenteric border of inflamed bowel – looks like teeth of a comb. Indicates active inflammation.

6.2 Fibrofatty Proliferation (Creeping Fat)

Pathognomonic for Crohn's disease. If you see it, the diagnosis is Crohn's. Full stop.

- Hypertrophy of mesenteric fat wrapping around the serosal surface of inflamed bowel segments.
- **How to identify on CT:** increased attenuation of mesenteric fat (-40 to -60 HU vs normal -100 to -120 HU), fat wrapping around the bowel (bowel embedded in a cushion of fat), separation of adjacent bowel loops.
- Only seen in Crohn's (transmural disease) – NOT in UC (mucosal disease).

> **TIP:** "Creeping fat is Crohn's until proven otherwise. It tells you the inflammation is transmural, the disease is chronic, and it helps identify exactly which segments are involved."

6.3 Extra-GI Manifestations on Imaging

- **PSC:** 10% UC, 5% Crohn's. MRCP: multifocal short-segment strictures with "beaded" bile ducts. CT: periportal edema, bile duct wall thickening. A new dominant stricture in PSC = cholangiocarcinoma until proven otherwise.
- **Sacroiliitis:** erosions, sclerosis, joint space irregularity of SI joints. Up to 20% of IBD patients. May be incidental on abdominal CT – mention it.
- **Gallstones (Crohn's):** terminal ileum disease causes bile salt malabsorption leading to cholesterol gallstones. Prevalence ~2x general population.
- **Renal stones (Crohn's):** bile salt malabsorption leads to free oxalate absorption causing calcium oxalate nephrolithiasis.
- **DVT/PE:** IBD patients have 2-3x increased VTE risk. Always check iliac veins and IVC.

> **PEARL:** "Terminal ileum disease in Crohn's has a cascade of metabolic consequences: bile salt malabsorption causes gallstones, and the resulting oxalate hyperabsorption causes kidney stones. One diseased segment, two types of stones."

6.4 Types of Colitis – The Differential

Ischemic Colitis:

- Watershed areas: splenic flexure (Griffiths' point, SMA/IMA junction) and rectosigmoid junction (Sudeck's point, IMA/internal iliac junction).
- Thumbprinting: submucosal edema causing polypoid indentations. Symmetric, circumferential wall thickening.
- Elderly patient + vascular risk factors + watershed distribution.

Infectious – C. diff:

- **Accordion sign:** alternating bands of fluid trapped between thickened, edematous haustral folds.

- Pancolitis pattern. Marked wall thickening (often >10-15 mm, more dramatic than IBD).
- Can cause toxic megacolon.

Infectious – TB:

- **Cecal predominance.** Closely mimics Crohn's.
- Distinguishing features: necrotic (low-density) lymphadenopathy, contracted thick-walled cecum, peritoneal involvement (omental caking, ascites).

Radiation Colitis:

- Confined to the radiation field. History is everything.
- Acute: wall thickening, hyperenhancement. Chronic: fibrosis, stricture, fistulae.

> **PEARL:** "Splenic flexure wall thickening in a 70-year-old with atrial fibrillation is ischemic colitis. In a 25-year-old, it is a very different story."

6.5 What Surgeons Need from Radiology in IBD

The best radiology report reads like an **operative roadmap.** Address:

1. **Active inflammation** – which segments, severity (wall thickness, enhancement, pericolonic changes)
2. **Strictures** – number, location, length, inflammatory vs fibrotic, upstream dilatation
3. **Fistulae** – number, type, course. Perianal fistulae = pelvic MRI.
4. **Abscesses** – size, location, drainability (>3 cm and accessible = IR drainage preoperatively)
5. **Skip lesion mapping** – complete map of all involved and uninvolved segments
6. **Mesenteric vascular anatomy** – especially if prior surgery or mesenteric vein thrombosis
7. **Extraintestinal findings** – stones, PSC, thrombosis, sacroiliitis
8. **Remaining bowel length** – critical for short bowel syndrome risk

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7 Pitfalls, Mimics & Miscellaneous

7.1 Mesenteric Nodes and Masses

- Always use coronal reformats. Node chains along mesenteric vessels are in their native plane on coronal.
- Round, heterogeneous, or necrotic nodes are more concerning than large reactive nodes with fatty hila.

7.2 Carcinomatosis

- Omental caking, peritoneal nodularity, scalloping of liver/spleen surface, ascites.
- Multiple transition points in SBO = think carcinomatosis.
- Can be subtle. Always look in the paracolic gutters, pouch of Douglas, and along the peritoneal surfaces.

7.3 Appendiceal Mucocele

CT Appearance:

- Well-defined, round/tubular cystic structure in the RIF at the expected appendiceal location.
- Low-attenuation contents (mucus). Appendiceal diameter >1.3 cm (normal <6mm).
- **Curvilinear mural calcification** in up to 50% – highly suggestive.

Why you CANNOT miss this:

- Rupture –> pseudomyxoma peritonei (PMP): mucin-secreting cells disseminate throughout peritoneal cavity. Progressive mucinous ascites, omental caking, bowel obstruction. Requires cytoreductive surgery + HIPEC.
- **Surgical implications:** avoid laparoscopic appendectomy (risk of iatrogenic rupture and peritoneal seeding). Open approach with careful handling. If frozen section shows malignancy, right hemicolectomy. Inspect peritoneal cavity and contralateral ovary.

> **TIP:** "A big, cystic, calcified appendix is NOT just a funny-looking appendicitis. It is a mucocele, and if the surgeon pops it, the patient may end up with pseudomyxoma peritonei. Call the surgeon BEFORE the operation and say: do not grab this with a laparoscopic grasper."

7.4 Internal Hernia

Background: After laparoscopic Roux-en-Y gastric bypass, mesenteric defects are created (Petersen defect and transverse mesocolon defect). Internal herniation occurs in 0.2-11%. Mortality rate 1.6% if missed.

Key CT Signs:

Sign	Description
Swirl sign	Rotation of mesenteric vessels/fat around an axis. Single best sign.
Clustered small bowel	Abnormal grouping of jejunal loops in LUQ or central abdomen
Mesenteric congestion	Engorged, edematous mesenteric vessels
SMV beaking	Tapering/compression of the SMV
Right-sided anastomosis	Jejunojejunostomy displaced to right of midline

In Pregnancy:

- Pregnancy is a **risk factor** – enlarging uterus displaces bowel and opens mesenteric defects.
- Presentation (pain, nausea, vomiting) is easily attributed to pregnancy itself, leading to **delayed diagnosis.**
- Consequences of delay: bowel ischemia, gangrene, perforation, fetal demise.

> **TIP:** "Pregnant woman with prior gastric bypass and acute abdominal pain = internal hernia until proven otherwise. Do not let concern about radiation delay imaging – missing this diagnosis can kill both mother and baby."

7.5 Epiploic Appendagitis

Torsion or spontaneous venous thrombosis of an epiploic appendage (small fat-filled peritoneal outpouchings along the colon).

Pathognomonic CT Appearance:

- **Fat-density oval** (1-4 cm) abutting the anterior colonic wall
- **Hyperattenuating ring sign:** thin hyperdense rim
- **Central dot sign:** thrombosed central vein (present in 30-78%)
- Surrounding fat stranding
- **No bowel wall thickening** (key distinction from diverticulitis)
- Usually sigmoid or cecum

Self-limiting – resolves in 1-2 weeks with NSAIDs alone. No antibiotics. No surgery.

> **TIP:** "Fat oval + hyperattenuating ring + no bowel wall thickening = epiploic appendagitis. Send the patient home with ibuprofen. Recognizing this saves an unnecessary operation."

7.6 Omental Infarct

CT Appearance:

- Large (>5 cm) cake-like heterogeneous fatty mass with internal streaky stranding.
- Between anterior abdominal wall and transverse/ascending colon. **Right-sided predominance.**
- **No hyperattenuating ring** (unlike epiploic appendagitis).
- Not adherent to colon wall (separated by fat cleft).
- Clinically mimics appendicitis or cholecystitis due to right-sided location.

Key Differentiators from Epiploic Appendagitis:

- Larger (>5 cm vs 1-4 cm)
- No ring sign
- In the omentum, not at the colonic surface

> **PEARL:** "Omental infarct is the big sibling of epiploic appendagitis. Right-sided location means it mimics appendicitis. Again, conservative management prevents an unnecessary operation."

7.7 Diverticulitis with Hepatic Abscess

Pylephlebitis (portal vein thrombophlebitis) from diverticulitis can seed the liver causing hepatic abscesses. **Always look for portal vein thrombus and gas** in the setting of diverticulitis with concurrent hepatic lesions.

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8 CT Fluoroscopy Procedures

8.1 CT vs Fluoroscopy for Fistula Assessment

CT provides cross-sectional detail of surrounding tissues but may miss small, tract-like fistulae that are better demonstrated dynamically.

Fluoroscopy shows real-time flow of contrast through fistulae but lacks the soft-tissue detail of CT for staging concurrent pathology (abscess, tumor).

Best approach: complementary. CT for anatomic overview and staging; fluoroscopy for dynamic fistula mapping.

Key Take-Home Points

1. **Coronal reformats are essential** – mesenteric vessels, nodes, obstruction transition points, and carcinomatosis are all best seen on coronal.
2. **The phase determines what you see** – portal venous for staging, arterial for bleeding/vascular mapping, delayed for ureters/bladder.
3. **Thin wall = arterial ischemia; thick wall = venous ischemia** – the single most important distinction in mesenteric ischemia.
4. **CTC is not a staging CT** – it finds polyps, but a separate contrast-enhanced CT is needed for staging.
5. **Diverticulitis vs sigmoid cancer** – 2-11% of complicated diverticulitis harbors underlying CRC. Always scope after resolution.
6. **Name the artery on CTA GI bleed** – IR and surgeons need a target.
7. **Mucocele = do not pop it** – communicate with the surgeon before any operation on a cystic appendiceal lesion.
8. **Epiploic appendagitis and omental infarct** are self-limiting mimics that save unnecessary surgery when correctly diagnosed.

9. **Every rectal cancer deserves an MRI** – the CRM drives the neoadjuvant decision.
10. **Always check the anastomosis** on follow-up imaging – anastomotic recurrence is commonly missed.