Acute abdomen in cancer patients: Role of imaging in a challenging clinical scenario

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Learning objectives

1. To review the gamut of etiologies presenting with an acute abdomen in cancer patients

2. To understand the unique clinical, diagnostic and management challenges posed by acute abdomen in oncology

3. Describe the common imaging manifestations and the role of imaging in the assessment of these patients

Background

Acute abdomen is a common presentation among cancer patients and is seen in up to 40% of cancer patients presenting to the emergency department [1]. Indeed acute life threatening abdominal emergencies including bowel obstruction, perforation and bleeding may be the first presentation of an underlying tumor. Advances in cancer treatment and the resultant increased life expectancy means that this is a frequently encountered situation for both clinicians and radiologists.

Causes:

Acute abdomen may be secondary to both neoplastic and non-neoplastic etiologies and can be multifactorial. Causes include:

- **Related to tumor**:
  
  Direct: mechanical effect of tumor, tumoral bleed, progression of tumor
  
  Indirect: systemic manifestations including immunosuppression, hypercoagulability

- **Secondary to treatment**: surgery related complications, side effects of chemotherapy/radiotherapy, marrow transplantation complications

- **Co existent unrelated causes**
Clinical and management challenges:

Apart from the wide range of possible underlying causes, clinical assessment is further complicated by the often atypical clinical features and delayed presentation. Immunosuppression may blunt many of the findings usually seen in a healthy population. Potentially any of the abdominal organs can be involved and the paucity of symptoms, clinical signs and laboratory evidence can make localization difficult with delay in diagnosis and subsequent treatment of the underlying condition.

Apart from the diagnostic issues, managing acute abdomen in oncology is also fraught with challenges. Identification of surgical and non-surgical candidates and proper selection of patients for emergent surgery is essential in the setting of high perioperative morbidity and often poor long term prognosis. A number of emergencies can be managed conservatively however selected causes of acute abdomen, most commonly bowel obstruction, perforation and acute bleeding would need intervention and prompt surgery in these scenarios is often life saving and associated with good short term outcomes.

Role of imaging:

Understanding diverse manifestations of this condition and the management implications is essential for the radiologist. Multimodality imaging, in particular CT plays a key role in identification of the underlying cause, mapping the extent of disease and triaging surgical and non-surgical candidates. Additionally imaging guided interventions including embolisation and percutaneous drainages play an increasingly important role in the non surgical management of these patients. Plain radiographs are often done as the initial examination for gastrointestinal causes such as obstruction and perforation. CT is the imaging mainstay allowing for both identification of cause and disease staging. Ultrasound, MRI and fluoroscopic studies can be useful in select cases with ultrasound being a useful first look tool in the pediatric population.

Findings and procedure details

We review the key imaging manifestations of acute abdomen in cancer patients across different systems with the underlying causes and management.
BOWEL:

- Most common cause of acute abdomen
- Include obstruction, perforation and infective/inflammatory conditions.

Intestinal obstruction:

- **Malignant bowel obstruction** defined as one of the following: clinical/radiological evidence of bowel obstruction (BO), BO beyond the ligament of Trietz, in the setting of a diagnosis of intraabdominal cancer or a non-intra-abdominal cancer with clear intraperitoneal disease.

- Common cause of acute abdomen, 3% per year and higher in colorectal (10-28% /yr) and ovarian cancer (20-50% /yr) [2,3]

- May be initial presentation

- Can be partial or complete

- Small bowel >> large bowel obstruction

**Causes**: can be due to mechanical or functional pathology

a) **Mechanical**: intraluminal (primary/secondary tumor, inflammatory causes); intramural (infiltration of bowel wall); extramural (extrinsic compression from masses, nodes, adhesions), intussusception

b) **Functional**: tumor infiltration of mesentery, pain medications, post surgery, paraneoplastic neuropathy

- **Gastric outlet and large bowel obstruction** - often due to malignant causes (Fig. 1 on page 14, Fig. 2 on page 14, Fig. 3 on page 14)

**Imaging**: 
1) Key clinical questions:

- Level of obstruction and cause
- Potential for strangulation / perforation
- Need for immediate operative intervention

2) Abdominal radiographs:

- Rapid, can be done bedside, can guide early management and imaging
- Findings: gaseous distension of the small or large bowel or bowel loops forming air-fluid levels on erect films; dilated small bowel loops with gasless colon indicates small bowel obstruction (SBO), presence of air fluid differential height in the same small bowel loop and a mean level width greater than 2.5 cm suggests high grade or complete SBO

3) Small bowel follow-through/ enterolysis/ contrast enema - not commonly done

4) CT:

- Superior to plain radiographs for identifying transition point, cause and adjacent anatomy, identifying closed loop/strangulation

- Findings:
  - Bowel wall thickening / soft tissue mass in bowel neoplasms (Fig. 1 on page 14, Fig. 2 on page 14, Fig. 3 on page 14), extrinsic masses/nodes (Fig. 4 on page 15)
  - Intussusception seen as target sign or a "loop within loop" pattern, with or without invaginated fat and mesenteric vessels (Fig. 5 on page 15)
  - If no identifiable mass, BO could be due to adhesions (Fig. 6 on page 16)
  - Can identify simple vs closed loop obstructions and complications like strangulation / perforation. This is important as closed loop obstructions and complications are surgical candidates while simple MBO initially managed conservatively

Management:

1. Conservative:
Decompression, fluid resuscitation, pharmacotherapy (scopolamine, octreotide)

2. **Surgical**:

- Associated with high morbidity (42%), mortality (5-32%) and recurrence rates (10-50%) [4]

- Consider surgery for closed loop obstruction, strangulation, simple BO worsening on conservative management

- **Malignant LBO - stenting, surgery**

**Bowel perforation**:

- May be **free** (where bowel contents spill into the abdominal cavity) and **contained** (if contiguous organs wall off the area)

- Often free in immunocompromised patients due to delayed presentation and patient's inability to localize infection

**Causes**:

1. **Tumor** - transmural perforation of bowel tumor (Fig. 7 on page 16, Fig. 8 on page 17) or invasion from adjacent malignancy (Fig. 9 on page 17), secondary to metastases

2. **Opportunistic infections** - CMV, candida, tuberculosis

3. **Neutropenic colitis**

4. **Ischemic colitis**, fecal impaction, diverticular disease in immunocompromised patients

5. **Secondary to chemotherapy** (including drugs causing pneumatosis) Bevacizumab (antiangiogenic agent), sorafenib, sunitib, capecitabine (Fig. 10 on page 18)

   - Incidence of perforation in patients undergoing bevacizumab-containing therapy is 0.9%-4% [5,6].

6. **Anastomotic leaks**:
• **Postoperative** anastomotic leaks occur in **first 3 months** after surgery (Fig. 11 on page 19)
• **Delayed perforation > 1 year** after surgery could be due to recurrent tumor or antiangiogenesis therapy (eg, bevacizumab)

**Imaging:**

• **Abdominal radiographs:** rapid initial investigation to look for free intraperitoneal air

• **CT:** Highly sensitive and can demonstrate small amounts of free air (Fig. 9 on page 17, Fig. 10 on page 18)

**Other CT findings** include:

• **Abscess or focal collection** of extramural fluid and/or air next to perforation site (Fig. 7 on page 16, Fig. 8 on page 17)
• Phlegmon/ inflammatory soft tissue mass/mesenteric stranding adjacent to site of perforation
• Abnormal segment of bowel with wall thickening or **pneumatosi**s
• **Leakage of luminal contrast** material at perforation site
• **Anastomotic leak findings** (Fig. 11 on page 19) are dehiscence near the surgical clips, increased fluid adjacent to the anastomosis site, colonic wall thickening, abscess formation and free intraperitoneal air. Mass and splaying sutures if related to recurrent tumor

**Management:**

• Broad spectrum antibiotics and usually operative management
• Delayed anastomotic leaks - if chemo related, treated by cessation of chemotherapy and conservative management.

**Bowel inflammatory/ infective changes:**

**Neutropenic colitis (Typhilitis):**

• Transmural inflammation of the cecum, proximal colon, and terminal ileum [7] seen in patients on chemotherapy, hematological malignancies and undergoing bone marrow transplant
• Characterised by **febrile neutropenia and abdominal pain,** particularly in the right iliac fossa
Typically symptoms occur 10-14 days after initiation of cytotoxic chemotherapy. Chemotherapy/leukemic infiltrates causes damage to the colon mucosa, and neutropenia leads to bacterial infection and necrosis. Pneumatosis and fluid or abscess formation may also be present. US may also be useful, especially in children.

Imaging:

- **Plain films**: nonspecific, findings include thickened dilated loops of bowel in the right lower quadrant, thumbprinting or localised pneumatosis intestinalis.

- **Ultrasound**: bowel wall thickening right lower quadrant, maybe useful in children (Fig. 12 on page 19)

- **CT**:
  - **Large bowel wall thickening** (mean thickness 7 mm) [8]; degree of wall thickening significantly correlates with the outcome of patients [9] (Fig. 12 on page 19, Fig. 13 on page 20)
  - **Right colon most commonly affected** (upto75%), isolated caecal involvement - 30% [9]
  - **D/D**: pseudomembranous colitis (PMC), infections, GVHD
  - Combined involvement of both large and small bowel more common in typhlitis
  - Degree of bowel thickening less in typhlitis (cf. PMC)
  - Pneumatosis intestinalis - 20% cases, more specific for typhlitis

Management: conservative, broad spectrum antibiotics, correction of cytopenias

Pseudomembranous colitis:
• **Infectious condition caused by immunosuppression**, mucosal damage from chemotherapy, and **superinfection with Clostridium difficile**.

• Clinical features: abdominal pain, nausea, and vomiting

**Imaging:**

• **Plain films**: may be normal or show non specific paralytic ileus; features of colonic edema such as haustral thickening and thumbprinting.

• **Ultrasound**: Moderate - severe colonic wall thickening, ascites

• **CT findings**: **Focal or diffuse eccentric or circumferential colon wall thickening** (mean 11-15 mm); degree of wall thickening more than other colitides [9] (Fig. 14 on page 20),

• **Marked mucosal enhancement**, target or halo sign, accordion sign in patients with bowel contrast

• Mild pericolic thickening, associated lymphadenopathy and ascites may be present

• **Stool culture or endoscopy** is used to confirm the diagnosis

**Treatment** - Antibiotic therapy, bowel rest with cessation of chemotherapy in severe cases

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**Acute Graft versus host disease (GVHD):**

• Occurs when **functionally competent T-lymphocytes are introduced into an immunocompromised recipient**

• Seen within the **first 100 days of allogenic bone marrow transplantation**

• Clinically significant GVHD occurs in **15-50% of adults** who receive an allogenic bone marrow transplant with skin, GI tract and liver the principally targeted organs.

• Presenting abdominal symptoms are non-specific

• **Imaging**:

**CT**: - **Small bowel continuous wall thickening** (Fig. 15 on page 21) may also be discontinuous

Large bowel involvement +/-, Mucosal contrast enhancement

Mesenteric stranding and engorgement of the vasa recta
**Extra-intestinal findings:** gallbladder wall thickening/ enhancement (peri-portal & peri-cholecystic fluid, hepatosplenomegaly.

Ultrasound can be done as initial study in the pediatric population and for hepatobiliary findings (Fig. 15 on page 21)

**Radiotherapy related bowel complications:**

**Small bowel:** - More radiosensitive than large bowel, fixed location of the terminal ileum makes it prone to radiation injury. Doses of approximately 50 Gy given over 6 weeks causes radiation enteritis with pelvic irradiation associated with a 5% risk for severe complications [10]

**Large Bowel** :- Cecum, rectum and sigmoid are most radiosensitive portions. Rectum and sigmoid most affected in RT for gynecologic and urologic tracts. Approximately 75% of patients who receive radiation to the pelvis develop symptoms of acute proctitis

- Underlying pathologic process is endarteritis obliterans, and compromise of the microvascular circulation; the vascular insult to the bowel wall is worsened by concurrent chemotherapy

**Imaging** :

i. **Small bowel imaging** - "thumb printing" seen in acute changes, not commonly done in acute setting

ii. **CT** :

- Enteritis seen as bowel wall thickening with a halo sign, serpentine appearance of the bowel, dilatation, mesenteric fat stranding, increased attenuation (from #100 to 20 HU) of the mesentery [11].

- May result in bowel obstruction secondary to colitis and post radiation strictures/adhesions (Fig. 16 on page 22), perforation, and fistulas.

- Look for pneumatosis and portal venous gas to confirm bowel ischemia and necrosis

iii. Radiation proctitis seen on CT as rectal wall thickening and perirectal fat stranding.

**Treatment** is usually supportive; surgery may be needed for complications like obstruction/ischemia/perforation
Other Inflammatory intestinal changes:

Bowel ischemia can be multifactorial (due to compression/invasion from malignant tumors causing vascular occlusion, bacterial proliferation associated with intestinal distension and chronic stasis, related to chemoRT, secondary to other infective/inflammatory colitides). Presence of pneumatosis, pneumoperitoneum, and pericolic collections suggest developing necrosis or perforation requiring urgent surgical evaluation. Other bowel related causes of acute abdominal pain seen in a normal population including appendicitis, diverticulitis, and inflammatory bowel diseases may also seen in the cancer population; some of these benign inflammatory conditions and neoplasia can have overlapping features or may co-exist (Fig. 17 on page 23, Fig. 18 on page 23).

VASCULAR CONDITIONS:

Includes intra-abdominal hemorrhage, vascular thromboses and occlusion.

Severe intraabdominal hemorrhage in cancer rare, but potentially fatal complication

Causes:

- Tumoral rupture:

  Hypervascular malignancies (Fig. 19 on page 23, Fig. 20 on page 24) eg hepatocellular carcinoma (HCC), renal cell carcinoma, and melanoma most common cause for spontaneous hemoperitoneum [12]. 10%-15% HCC present to the emergency with abdominal hemorrhage. Others include spontaneous splenic rupture in lymphoma/leukemia.

  Risk factors: Large size of mass, peripheral or subcapsular location, increased vascularity

- Secondary to coagulopathies for eg leukemia especially post chemotherapy (Fig. 21 on page 25)

- Tumoral invasion

Imaging: Imaging mainstays are CT and angiography

- High attenuation ascites (30-45HU) in acute hemoperitoneum, fluid levels, subcapsular or intraabdominal hematomas on unenhanced CT
• Clotted blood has higher attenuation of 45-70 HU, sentinel clot may indicate the source of bleeding
• Active extravasation of contrast (Fig. 19 on page 23, Fig. 20 on page 24, Fig. 21 on page 25, Fig. 22 on page 25) material on contrast enhanced CT identifies site of bleed and indicates ongoing bleeding.
• Can be confirmed on angiography (embolization can be done in the same sitting) (Fig. 20 on page 24, Fig. 22 on page 25)

**Treatment** - imaging guided embolization or emergent surgical exploration

**Hepatic Veno-occlusive Disease (sinusoidal obstruction syndrome):**

• Vascular congestion of the liver caused by sinusoidal injury
• More commonly associated with stem cell transplantation and high-dose chemotherapy in patients with leukemia; also seen in conventional chemotherapeutic agents (for eg oxaliplatin)
• Seen in 10%-60% of patients undergoing stem cell transplantation

**Clinical features:** Abdominal pain, tender hepatomegaly, jaundice, hepatic failure. May mimic GVHD.

**Imaging:**

**US:** enlargement and heterogeneity of the liver, periportal edema, ascites, gallbladder wall thickening, Doppler findings of reduced hepatic venous flow, reversed portal venous flow.

**CT:** Hepatic heterogeneity with geographical hypodensities (Fig. 23 on page 26), periportal edema, ascites, narrowing of the hepatic veins. Splenomegaly and varices indicate portal hypertension

**D/d:** GVHD - hepatic vein narrowing, periportal edema, ascites more in HVOD; small bowel thickening in GVHD. Consider biopsy for differentiating

**Treatment:** No effective treatment, pharmacotherapy

**HEPATOBIILIARY AND PANCREATIC CONDITIONS:**

**Pancreatitis:**
**Secondary to chemotherapy** (asparaginase, ifosfamide, paclitaxel, cisplatin, vinorelbine, cytarabine, and tretinoin) [13], and surgery (cytoreductive surgery and intraperitoneal chemotherapy).

- Asparaginase-associated pancreatitis has a prevalence of 2%-16% [13,14] (Fig. 24 on page 27).

- Symptoms usually hours after chemotherapy but may be delayed

- **Imaging** - diffuse or focal pancreatic swelling, areas of low attenuation or diffuse edema, peripancreatic fat stranding (Fig. 24 on page 27) and peripancreatic fluid. CT may be normal with lab evidence of pancreatitis.

- **Treatment** - supportive, alternate chemotherapy can be considered.

**Hepatobiliary** :-

**Pyogenic and fungal abscesses** may be seen secondary to immunocompromised state. Sometimes hepatic abscesses can be seen as an unrelated cause of acute abdomen in a patient with cancer (Fig. 25 on page 27). Treatment consists of antibiotics and percutaneous drainage. **Cholangiolar abscesses** (Fig. 26 on page 28) and **biliary inflammation** (cholangitis / cholecystitis) can be seen as complication for stenting for biliary obstruction most commonly in hepatobiliary malignancies. **Bowel perforation may be seen as complication of ECR** (Fig. 27 on page 28). Cholecystitis also may be due to an unrelated cause, additionally GVHD and parenteral nutrition predispose to cholestatis and calculi/sludge formation.

CT in hepatic abscesses shows single or multiple hepatic hypodensities (Fig. 25 on page 27, Fig. 26 on page 28) with metastases being the most common differential; presence of air within the lesion and "cluster sign" are useful clues. Cholecystitis is seen as gall bladder wall thickening, pericholecystic fluid and stranding. Features of complicated cholecystitis and perforation should be looked for as these may require surgery or urgent cholecystotomy.

**Miscellaneous conditions** :

Other organs systems such as **musculoskeletal** (Fig. 28 on page 28) and **genitourinary** (Fig. 29 on page 29) can also be involved with infections and urinary obstruction being the most common causes of acute abdomen.
**Images for this section:**

![Image](image1)

**Fig. 1:** 85 year old presenting with acute onset abdominal pain and vomiting. (A) Plain abdominal radiograph showing a massively distended stomach (arrows). (B) Coronal section of contrast enhanced CT scan showing stenosing tumour of the gastric antrum causing gastric outlet obstruction (arrow). (C) Plain radiograph showing gastric decompression post luminal stenting (arrow).

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![Image](image2)

**Fig. 2:** 50 year old male presenting with acute onset abdominal pain. (A) Axial section of contrast enhanced CT scan showing dilated small and large bowel loops in keeping with intestinal obstruction. (B and C) Axial and coronal sections of contrast enhanced CT scan showing circumferential mural thickening with luminal stenosis of sigmoid colon in keeping with a primary carcinoma (arrows).

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**Fig. 3:** Another example of a colonic malignancy with initial presentation as acute abdomen. A 64 year old male presenting with an "apple core" sigmoid colonic mass (thick arrows) on axial (A) and coronal (B) sections of contrast enhanced CT with upstream large bowel obstruction (thin arrows). The patient later underwent anterior resection of the sigmoid mass

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**Fig. 4:** 67 year old male. Case of gallbladder carcinoma, treated with primary resection, Presented with acute abdominal pain. (A - C) Axial and coronal sections of contrast enhanced CT scan showing peritoneal deposits in abdomen and pelvis (thick arrows) suggestive of peritoneal disease. Also there is a peritoneal deposit at the rectosigmoid region (C) causing extraluminal compression of sigmoid colon and resultant bowel obstruction (thin arrows) (A).

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**Fig. 5:** 87 year old male presenting with acute onset abdominal pain. (A and B) Axial and coronal sections of contrast enhanced CT scan showing ascending colon intussusception. The intussuscipiens (thin arrow) consists of a primary cecal tumour while the intussusceptum is the ascending colon (thick arrow).

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**Fig. 6:** Intestinal obstruction secondary to adhesions. 52 year old male post proctocolectomy and local radiotherapy presented clinically with intestinal obstruction. Gastrograffin follow through (A) showed several dilated small bowel loops with delayed small bowel transit time. Axial (B) and coronal (C) sections of contrast enhanced CT showed loop of small bowel (arrows) in the pelvis that was the transition point with no associated mass or wall thickening suggesting adhesions as the underlying cause.

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**Fig. 7:** 84 year old male presenting with acute abdominal pain. (A and B) Axial sections of contrast enhanced CT scan showing primary tumour of ascending colon with mural breach and pericolonic abscess formation (arrows).

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**Fig. 8:** 91 year old female presenting with acute onset abdominal pain. (A and B) Axial and coronal sections of contrast enhanced CT scan showing primary tumour of sigmoid colon with perforation (thick arrows), invasion into left psoas muscle with resultant abscess formation (thin arrows). (C) CT guided drainage of psoas abscess was performed.

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Fig. 9: 60 year old female, known case of endometrial carcinoma involving the left ovary with disease progression resulting in sigmoid invasion and resultant perforation. Initial axial contrast enhanced CT (A) and axial DWI MRI (B) demonstrate the endometrial (thick arrow) and left ovarian (thin arrow) masses. The patient subsequently deteriorated. Contrast enhanced CT done shows pneumoperitoneum (arrow) and peritoneal disease (arrow) on axial sections (D, E). Coronal section (F) shows the uterine mass abutting the sigmoid colon (arrow); few tiny air locules adjacent to the sigmoid indicate the site of perforation.

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Fig. 10: 65 year old female. Case of rectal adenocarcinoma treated with low anterior resection and neoadjuvant chemoradiotherapy [chemotherapeutic agent XELODA (Capecitabine)]. Presented with right sided abdominal pain. (A and B) Axial and coronal sections of contrast enhanced CT scan showing diffuse inflammatory mural thickening of cecum and ascending colon (black arrows) with free air locules in the vicinity (white arrows) in keeping with colonic perforation.

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Fig. 11: 67 year old male. Case of sigmoid adenocarcinoma treated with primary resection, presented with acute abdominal pain 2 weeks post surgery. (A) Axial section of contrast enhanced CT scan showing large air containing abdominal collections (arrow). (B) Coronal section of contrast enhanced CT showing connection between descending colon anastomotic site and pneumoperitoneum suggestive of anastomotic leak (arrow). (C) CT guided drainage of collections was performed.

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Fig. 12: 10 year old female with acute lymphoblastic lymphoma with typhilitis and classic clinical features of febrile neutropenia and right abdominal pain. Ultrasound (C) showed diffuse thickening of the right colonic wall (arrow). Axial (B) and coronal (C) sections of contrast enhanced CT also show diffuse circumferential right colonic wall thickening (arrow) with mucosal enhancement and submucosal edema. Mild pericolonic inflammatory changes also seen.

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Fig. 13: 14 year old female with newly diagnosed acute myeloid leukemia and prolonged febrile neutropenia presenting with central abdominal pain with diarrhea. Axial (A) and coronal (B) sections of the contrast enhanced CT study show diffuse wall thickening and oedema of the caecum extending to the ascending and transverse colon (arrows) with pericolonic fat stranding.

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Fig. 14: 73 year old male. Case of lymphoma treated with 5 cycles of CEPP (cyclophosphamide, etoposide,procarbazine and prednisone). Presented with acute abdominal pain and diarrhoea. E-Coli and Clostridium Difficile toxin tests were positive. (A and B) Axial and coronal sections of contrast enhanced CT scan showing long segment circumferential mural thickening of descending and sigmoid colon with intense mucosal enhancement and surrounding fat stranding (arrows), compatible with pseudomembranous colitis.

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Fig. 15: 16 year old male patient; known case of AML post allogenic bone marrow transplant with skin changes of GVHD and acute abdominal pain. CECT axial (A) and coronal (B) sections show diffuse small bowel (thin arrow) mural thickening, mucosal enhancement and submucosal edema with similar changes in the large bowel (thick arrow). Ultrasound (C) shows diffuse thickening and edema of the gall bladder wall with sludge (arrow).

Fig. 16: 59 year old male with prior sigmoid carcinoma with low anterior resection and chemoRT, was admitted for symptoms of intestinal obstruction with vomiting diarrhoea. CECT axial (A) shows diffuse thickening of the colon in the pelvis in keeping with post radiation changes. Mild diffuse thickening seen in the small bowel loops which are dilated (arrows) (B) with the coronal image (C) showing an abrupt zone of transition along the jejunal bowel loops in the region of the pelvis (arrow) probably due to underlying adhesions. Patient was initially treated conservatively but later underwent adhesiolysis.
and surgery confirmed obstruction due to dense post RT fibrous adhesions. Post RT changes were also seen in the small and large bowel

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**Fig. 17:** 62 year old female with acute onset right iliac fossa pain with CECT axial (A) and coronal images (B, C) showing a dilated thick walled appendix (arrows). Surgery showed a thickened inflammed pus filled appendix with perforation. Histopathology showed invasive moderately differentiated adenocarcinoma of the appendix against background inflammatory changes

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**Fig. 18:** 66 year old presenting with acute abdominal pain. (A and B) Axial and coronal sections of contrast enhanced scan showing short segment mural thickening of sigmoid colon (arrows) with surrounding fat stranding in the presence of colonic diverticula, suspicious for diverticulitis. Surgical findings were of diverticulitis superimposed on primary sigmoid adenocarcinoma.

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Fig. 19: 60 year old male with acute onset bleeding per rectum and lower abdominal pain. Axial section CECT shows an enhancing soft tissue jejunal mass (thin arrow) in the left lower abdomen with contrast extravasation in keeping with bleeding (thick arrow). This was confirmed to be a GIST on surgery and histopathology.

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Fig. 20: 62 year old male, a known hepatitis B virus carrier, presenting with acute onset abdominal pain. (A) Axial section of contrast enhanced CT scan showing right hepatic lobe heterogeneous mass with capsular breach, contrast pooling (thick arrow) and hemoperitoneum (thin arrow) suggestive of a ruptured hepatocellular carcinoma with active peritoneal hemorrhage. (B) Catheter angiogram of hepatic artery proper showing active arterial hemorrhage at the periphery of tumour (arrow) which was then embolized.

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Fig. 21: 11 year old female patient with known acute lymphoblastic leukemia and acute onset bleeding per rectum and abdominal pain. CT angiogram showed active hemorrhage at the splenic flexure (coronal, B, thick arrow) with background diffuse bowel wall thickening (thin arrows).

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**Fig. 22:** 62 year old female with pancreatic carcinoma and prior Whipple's operation and chemotherapy who subsequently developed recurrence in the surgical bed. She presented to the emergency with acute onset gastrointestinal bleed. CT angiogram (A) showed severely attenuated celiac axis with massive active bleeding into duodenum noted from a likely eroded common hepatic artery/hepatic artery (arrows), treated by coil embolisation (C) (arrow)

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**Fig. 23:** 65 year old male. Case of non small cell lung carcinoma treated with chemotherapy (cisplatin/carboplatin). Presenting with abdominal tenderness, worsening transaminitis and conjugated hyperbilirubinemia. (A) Axial section of contrast enhanced CT scan showing heterogeneous enhancement of liver parenchyma (arrow) suggestive of hepatic sinusoidal obstructive syndrome. (B and C) Ultrasound done a few days later
shows complete thrombosis of portal veins likely due to poor antegrade flow and stasis (arrows).

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**Fig. 24:** 24 year old male patient with ALL on a chemotherapy regimen that included L-asparaginase; presented with central abdominal pain. CECT axial (A) and coronal sections (B) show prominent fat stranding and inflammatory changes (arrows) in the right anterior pararenal space centered around the duodenum and pancreatic head, extending inferiorly along the right retroperitoneum. The pancreas was normal apart from mild bulkiness of the pancreatic head. Serum amylase and lipase levels were elevated confirming the diagnosis of L-asparaginase related pancreatitis.

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**Fig. 25:** 56 year old female presented with fever and abdominal tenderness. (A) Axial section of contrast enhanced CT scan showing right hepatic lobe hypodense lesions compatible with abscesses (black arrows). (B and C) Axial and coronal sections of contrast enhanced CT scan showing a polypoidal luminal mass within the transverse colon (white arrows).

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Fig. 26: 48 year old female with cholangiocarcinoma and biliary stenting. CECT axial (A) shows a rim enhancing hypodensity and biliary stent (B), in keeping with cholangiolar abscess which later underwent imaging guided drainage

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Fig. 27: 56 year old male patient with painless obstructive jaundice, diagnosed as periampullary carcinoma on ERCP and biopsy. Post ERCP he complained of increased abdominal distension and pain; and plain radiograph (not shown) revealed pneumoperitoneum. This was confirmed on CECT (axial A, coronal B) which showed extensive pneumoperitoneum (thick white arrow) and pneumoretroperitoneum (thin white arrows) with a small amount of mediastinal emphysema (short black arrow) in keeping with post ERCP perforation with possible site of perforation at second part of duodenum (long black arrow). The patient underwent emergency Whipple's operation

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Fig. 28: 34 year old female with Acute Myeloid Leukemia. Presented with right lower abdominal pain and fever, mimicking symptoms of appendicitis. (A) Axial sections of contrast enhanced CT scan of abdomen and pelvis shows right retroperitoneal fat stranding suggesting some inflammatory change (arrow). (B and D) Coronal sections of contrast enhanced MRI abdomen and pelvis further show abnormal enhancement involving the right iliopsoas muscles with abscess formation (arrow). (C) CT guided drainage of right retroperitoneal abscess was performed.

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Fig. 29: Ectopic left testes with torsion secondary to neoplasm in a 28 year old male patient who initially presented with acute lower abdominal pain. CECT axial (A, B) and coronal (C) showed a heterogeneous low density pelvic mass (thin arrows) with surrounding high density fluid. The left testicular vessels formed the vascular pedicle of this mass and had a swirled appearance (not shown) in keeping with torsion. The left spermatic cord was not seen (thick arrows indicate the right spermatic cord) and an empty left scrotal sac was confirmed clinically. Findings were further confirmed on surgery; excision of the intra-abdominal testicular tumor was done and histology was seminoma.

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Conclusion

Acute abdomen is a unique clinical challenge in oncology. With advances in therapeutic options improving patient survival this is a common clinical scenario Imaging plays a central role in the diagnosis of cause and guiding therapy decisions with a growing role for imaging guided interventions. Radiologists should be aware of the imaging spectrum in these patients as early diagnosis and appropriate treatment is the key to improving patient outcome.

Personal information

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