REVIEW

Imaging of abdominal and pelvic infections in the cancer patient

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Abstract

Infections are the most commonly encountered complications in patients with cancer. The classical signs and symptoms of infections are often not present in this patient population, which makes the diagnosis more challenging. Host factors play a major role in the development and prognosis of infections in cancer patients; these can be related to the underlying type of malignancy (solid organ versus hematological), tumor burden, anatomic obstruction, altered integrity of barriers (skin or mucosa), treatment-related factors (from chemotherapy, radiation treatment, surgery, interventional procedures, and/or medical device placement) and the degree of immunosuppression. This article reviews common, as well as less common, imaging manifestations of infections and their potential mimics in the abdomen and pelvis in cancer patients and discusses their differentiating features, with the role of imaging in various organs in the abdomen and pelvis taking into consideration relevant clinical background information and the main risk factors.

Keywords Infections · Cancer · Abdomen · Pelvis · Gastrointestinal · Genitourinary

Introduction

Approximately 40% of the U.S. population develops cancer at some point in their lifetime, with over 1.8 million new cancer cases estimated to be diagnosed in 2020 [1]. There has been a remarkable 29% decline in cancer mortality in the U.S.A. between 1991 and 2017, with a relatively stable cancer incidence in women and a slight decline in cancer incidence in men, primarily due to a decrease in smoking [2]. Infections pose a higher risk of morbidity and mortality in cancer patients, both directly due to their immunocompromised state, and indirectly by delaying cancer treatment and

impacting long-term outcomes. Imaging plays an important role in the management of patients with suspected or known cancer, with substantial contributions related to screening, diagnosis, staging, evaluating response to therapy, and surveillance, as well as aid in detection of various complications, with infection being the most common [3]. Impairments to the immune system, mediated either by the tumor or therapies, predispose cancer patients to infection, potentially leading to serious outcomes [4]. The current coronavirus pandemic may serve as an example, with 2–3 times increase in COVID-19 related fatality rates in cancer patients [5]. The purpose of this article is to review the underlying risk factors and mechanisms of infections in patients with various malignancies and to present the variety of imaging manifestations of abdominal and pelvic infections in cancer patients.

Risk factors for infections in cancer patients

There are numerous risk factors that play a substantial role in the development of abdominal and pelvic infections in cancer patients. These can be divided into two main categories: risk factors that are related to tumor burden itself and those related to cancer treatments and interventions

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Table 1 Main risk factors for infection in cancer patients

Infection risk factors in oncology patients	Quick facts
Barrier injury	Disruption of skin and mucosa causing translocation of pathogens into the blood stream and sterile tissues
Chemotherapy, neutropenia, and immunosuppression	Most encountered in hematologic malignancies, specifically after bone marrow transplant
Obstruction	In solid tumors (carcinomas and sarcomas) frequently causing genitourinary and hepatobiliary infections
Vascular occlusion	Vascular invasion by solid tumors or bland thrombus due to hypercoagulable states namely in pancreatic and ovarian cancer
Radiation-induced damage	Causes inflammation, skin and mucosa barrier injury, small vessel sclerosis, fibrosis, and fistula formation
Medical device infection	Prime example is blood stream infections of vascular catheters caused by skin colonizers
Surgery	Can be major infections with high morbidity such as anastomotic bowel leaks, or minor infections such as surgical wound and urinary tract infections

(Table 1). Often, patients have a combination of risk factors at the time of exposure to infection. An increased risk of infections (influenza, gastroenteritis, hepatitis, and pneumonia) has been recently reported in cancer patients even before the diagnosis of cancer is made, indicating a more complex relationship between infections, immune suppression, and cancer formation [6].

Solid organ versus hematologic malignancies

There are basic differences in mechanisms predisposing to infections with solid organ malignancies when compared to hematologic malignancies (Table 2). In solid organ malignancies such as carcinomas and sarcomas, most patients are not substantially immunosuppressed (in contrast to hematologic malignancies) and do not experience prolonged neutropenia [3, 7].

In hematologic malignancies including lymphoma, chronic lymphocytic leukemia (CLL), and acute myeloid leukemia, neutropenia often develops due to either bone marrow infiltration by the tumor or effects of chemotherapy. Other immune system alterations also present in patients with hematologic malignancies, including decreased T-cell mediated immunity and hypogammaglobulinemia in patients with CLL [8].

Disruption of anatomic barriers

Skin and mucosal surfaces are a cornerstone defense mechanism for the body against infections. Cancer can directly

Table 2 Differentiating features of solid organ and hematologic malignancies and related infectious pathogens

	Hematologic malignancies	Solid organ malignancies
Malignancy incidence	Less common (leukemia, lymphoma, multiple myeloma)	More common (carcinoma, sarcoma)
Risk factors for infection	Severe prolonged neutropenia from chemotherapy and immunosuppression Altered permeability in mucosal barrier injury from cytotoxic chemotherapy	Multifactorial: obstruction from tumor, disruption of natural barriers
Distinctive entities	Febrile neutropenia Blood stream infections (bacteremia, candidemia) Neutropenic enterocolitis	Ascending cholangitis Abscesses (hepatic, renal, prostate) Bowel obstruction and necrosis, perforation Fistulas Obstructive uropathy Surgical site infections
Most common pathogens	Enterobacteriaceae Cytomegalovirus Invasive aspergillosis[a] Candida	Methicillin-resistant *S. aureus* *Pseudomonas aeruginosa* *Clostridium difficile* Extended spectrum beta-lactamase-producing organisms e.g. *Klebsiella pneumonia* Vancomycin-resistant *enterococci* Polymicrobial infections[b,c]

[a]Ruiz Camps [13]

[b]Sutton [1]

[c]Rolston [3]

disrupt these barriers by direct invasion, such as in cases of inflammatory breast cancer and ulcerative gastrointestinal tumors, or by therapies that either damage these barriers (such as catheter placement, radiation therapy) or affect their healing response (chemotherapy).

Mucosal barrier injury is more common in hematologic malignancies, particularly after bone marrow transplant (BMT), and typically presents with general gastrointestinal symptoms including nausea, vomiting, diarrhea, and abdominal complaints [9].

Alterations in permeability and loss of the epithelial surface are the principal characteristics of mucosal barrier injury seen with neutropenic enterocolitis, graft-versus-host disease (GVHD), veno-occlusive disease, and systemic infections. Mucosal barrier injury occurs in 4 phases (Fig. 1) [10].

Chemotherapy, neutropenia, and immunosuppression

Neutropenia, defined as an absolute neutrophil count of < 1500 cells per mm^3, develops in the majority of cancer patients (particularly those with hematologic malignancies) and is most commonly due to chemotherapy effects [11]. Additional causes of neutropenia in cancer patients include bone marrow infiltration by the tumor, irradiation of multiple red marrow sites, and myelosuppressive treatment. Severe neutropenia is defined as a cell count of < 500 cells per mm^3, and treatment is required when neutropenic patients are febrile [11]. With prolonged periods of febrile neutropenia and frequent antimicrobial therapies, patients with hematologic malignancies have resident flora that is often multidrug-resistant [12]. The most common pathologies of the lower digestive tract are enteric gram-negative bacillary infections (e.g., *Escherichia coli*, Klebsiella

species, *Pseudomonas aeruginosa*) and neutropenic enterocolitis caused by *Staphylococcus aureus*, and Clostridium species [1, 3, 12, 13]. These immunocompromised patients are also susceptible to serious viral infections and invasive fungal infections, namely those due to Candida and Aspergillus, and result in significant morbidity and mortality in patients with prolonged neutropenia such as those following BMT [14–16] (Fig. 2). Imaging plays an essential role in the detection of these infections and their complications.

## Obstruction due to tumor progression and vascular occlusion

Obstruction often complicates advanced solid tumors and is a predisposing factor for infections, as it leads to post-obstructive infectious processes, perforation, and abscess formation. Bowel obstruction by either the primary malignancy or metastatic deposits can be challenging to manage as it can be complicated by bowel ischemia, perforation, fistulas, peritonitis, and abdominopelvic abscesses, typically polymicrobial. Hepatobiliary obstruction can result in recurrent cholangitis and hepatic abscesses. Urinary obstruction may complicate advanced gynecologic or colorectal malignancies, and hydronephrosis can progress to pyonephrosis, pyelonephritis, and renal abscesses. Urethral obstruction, for example by prostate cancer, can result in prostatitis and prostatic abscesses.

Vascular occlusion can cause organ ischemia and poor circulation, which facilitates infection. Peripheral vascular insufficiency that leads to infected ulcers is a general example of an infection related to compromise in arterial flow and can result in complications such as abscess formation, osteomyelitis, and limb loss. Vascular occlusions, more commonly venous, can result from direct tumor extension to vessels, mass effect on underlying vascular structures (in
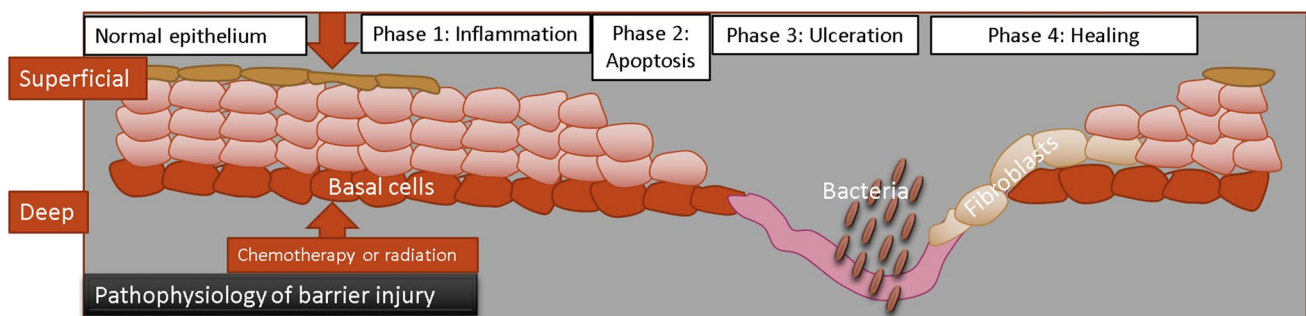
Fig. 1 Diagram of mucosal barrier injury. In phase I, cytotoxic drugs and/or irradiation cause activation of several cellular transcriptional pathways resulting in the induction of increased proinflammatory cytokines. The epithelial cells remain intact. In phase II, the epithelial phase, cells stop dividing and undergo apoptosis. This phase coincides with neutropenia. In phase III, necrosis and ulcerations

occur, enabling the resident microbial flora and their endotoxins to translocate into the bloodstream. Additional risk factors for infection in phase III are impaired local defenses and low levels of secretory IgA. In phase IV, healing is mediated by the action of naturally occurring substances such as trefoils and growth factors
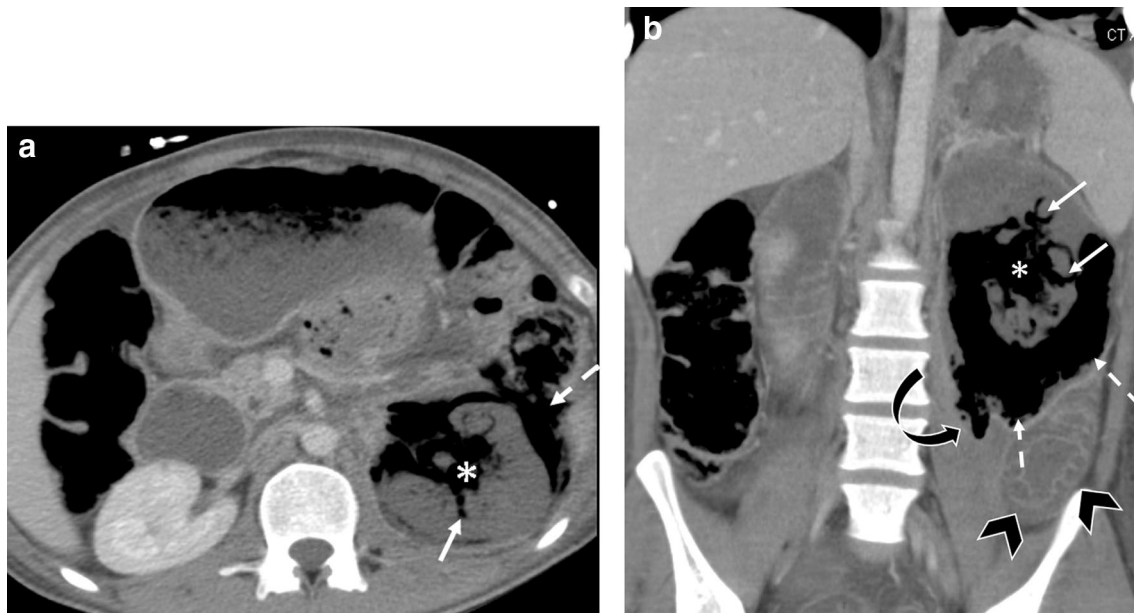
Fig. 2 Angioinvasive mucormycosis in a 28-year-old man with acute myelogenous leukemia and severe neutropenia. **a** Axial and **b** reconstructed coronal intravenous contrast enhanced CT images demonstrate diffuse left renal necrosis with emphysematous pyelonephritis manifested by absent left renal perfusion, gas in the left renal parenchyma (solid arrows), collecting system (asterisk) and extensive gas in the perirenal space (dashed arrows). There is also evidence of extension of gas into the retroperitoneum (curved arrow). Involvement of the descending colon with wall thickening and mucosal hyperemia is noted (arrowheads in **b**)

cases of solid tumors), or due to bland thrombus from hypercoagulable states (particularly in cases of pancreatic, gastric and ovarian cancers) [17].

Radiation-induced injury

A range of 50% to 70% of cancer patients receive radiation therapy sometime along the course of their cancer care [18, 19]. Radiation-induced injury leads to mucosal barrier injury, as the rapidly dividing cells in the skin and the mucosal lining of the gastrointestinal tract are quite radiosensitive. The extent of injury depends on the dose and the radiation field; and destruction of mucosal barriers and skin predisposes to the entry of infectious pathogens into the body and bloodstream. Radiation injury to the gastrointestinal tract has significant morbidity that affects the quality of life of cancer patients, and can often be the limiting factor for the dose of radiation that patients can tolerate [20]. The hallmark of acute radiation injury is inflammation, whereas that of chronic radiation injury is transmural fibrosis and vascular sclerosis [20]. The most common manifestations of acute radiation injury are mucositis (in 90% of patients with concurrent chemotherapy), esophagitis and esophageal dysmotility (grade 2–3 injuries in 46% with concurrent chemotherapy), colitis (50%), and anorectal complications (in up to 75%) [20]. Chronic complications associated with radiation injuries, such as stricture or fistula formation, can lead to obstruction, stasis, and viscus perforation, and commonly

superinfection and/or abscess formation (Fig. 3) [21]. Some of the radiation-induced changes can be readily recognized in imaging. For example, small bowel wall thickening and mucosal hyperenhancement are predominant initial imaging findings of radiation-induced bowel injury, whereas luminal narrowing is the most common feature of chronic radiation enteropathy. It is important to note that the differentiation of bland radiation-induced injury from superinfection may not be always possible due to the overlap of imaging findings. Another potential mechanism of chronic radiation-induced injury that may predispose to infection is on the basis of tissue hypoperfusion and hypo oxygenation associated with fibrosis and vascular sclerosis [22]. In addition to the radiation dose, the risk of radiation-induced injury depends on many factors such as body habitus, smoking status, pre-existing vascular disease, inflammatory bowel disease, collagen vascular diseases, human immunodeficiency virus infection (which is thought to increase radiosensitivity), and genotypical variations (Fig. 4) [20].

Medical device infection

Medical devices, such as vascular access catheters, have become widely utilized in the management of cancer patients. These devices can be the vehicle introducing infectious pathogens via disruption of anatomic barriers and can also complicate the management of these infections, necessitating the removal of such devices. The risk of infection
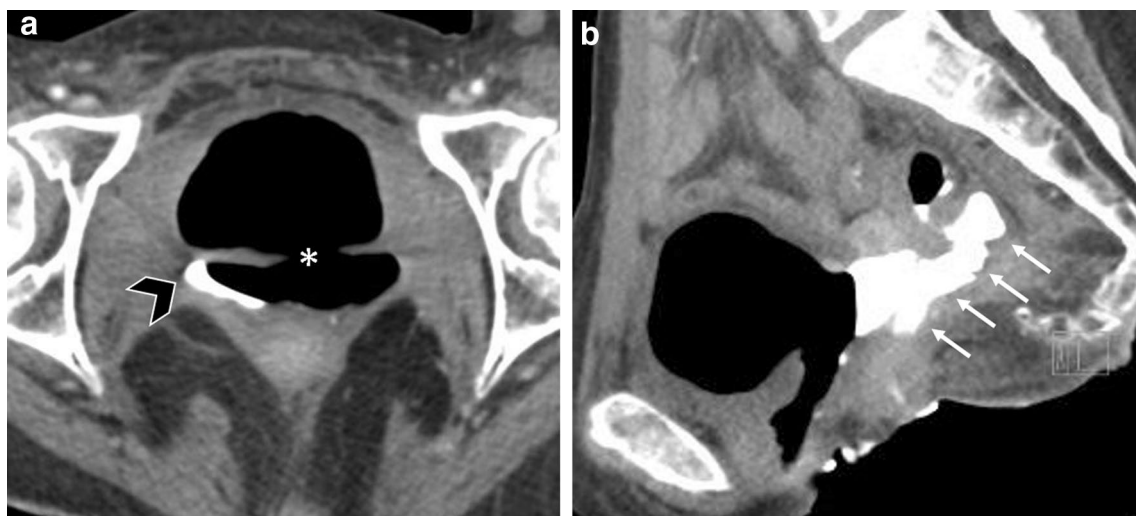
Fig. 3 Rectovaginal and vesicovaginal fistulas complicating radiation therapy in a 41-year-old woman with cervical cancer who presented with a large amount fecalous discharge from the vagina and bladder. **a** Axial and **b** sagittal reconstructed intravenous contrast enhanced CT images demonstrate dense contrast from prior enema in the vagina (arrowhead in **a**) and colon (arrows in **b**) consistent with a rectovaginal fistula. Note a large defect in the posterior bladder wall (asterisk) consistent with a vesicovaginal fistula. The patient has been managed with urinary diversion via placement of bilateral percutaneous nephrostomy tubes, and colostomy
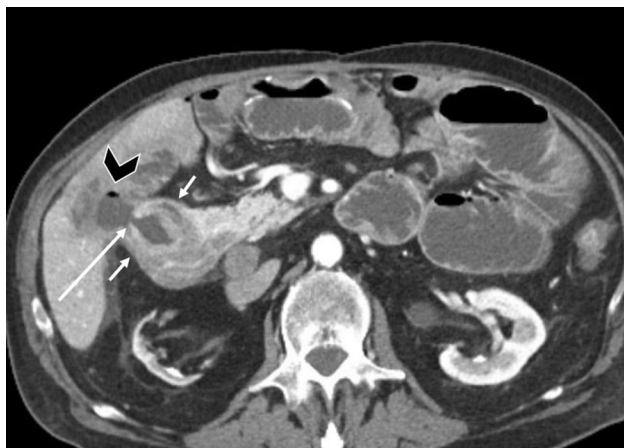
Fig. 4 Acute radiation enteritis in a 74-year-old woman with sigmoid adenocarcinoma and solitary liver metastasis who underwent hepatic radiation treatment (dose: 5000 cGy). Patient presented 1 week after completing radiation with abdominal pain, nausea, and vomiting. Axial intravenous contrast-enhanced CT image demonstrates a focal area of duodenal wall thickening with associated submucosal edema and stratification due to mucosal and serosal hyperenhancement (short arrows). This was complicated by fistulous communication (long arrow) between the duodenum and the necrotic cavity from radiated metastatic adenocarcinoma, which subsequently became a liver abscess (arrowhead). Note that only the part of the duodenum which was in the radiation field was affected

Fig. 5 Infectious complication of a gastrostomy tube in a 68-year-old man with glioblastoma multiforme post gastrostomy tube placement who developed fever and abdominal pain. Axial CT image with intravenous contrast demonstrates a percutaneous gastrostomy tube inadvertently placed in the left hepatic lobe (arrowhead) with fluid and gas collection in the adjacent liver consistent with polymicrobial hepatic abscess (arrows) and perihepatic complex fluid collection (asterisks) with enhancing walls (curved arrow) consistent with intraperitoneal abscess

with medical devices depends on a number of factors the most important of which is immunosuppression [23]. The most common pathogens in vascular catheter infections are skin colonizers, namely Staphylococcus species.

Gastrointestinal and abdominal wall devices such as percutaneous gastrostomy tubes can be complicated by superficial infections along the skin or abdominal wall muscles

or deeper infections such as peritonitis and abdominopelvic abscesses (Fig. 5). Peritonitis can also be caused by surgical catheters and shunts. Biliary stent-related events such as cholangitis and recurrent obstruction can be seen in more than 40% of patients [24]. Solid tumors, particularly those related to gynecologic malignancies, may require internal ureteral stents, surgical diversion, or percutaneous nephrostomy tubes, which increase the risk of acute and chronic pyelonephritis and bacteremia [25, 26]. Multi-modality imaging plays a substantial role in the assessment of these complications and provides a means for medical device surveillance.

Surgical procedures

Surgical site infections occur in 8% of cancer patients [27]. These include major infections that lead to complications such as anastomotic bowel leaks and biliary leaks and require additional surgeries, as well as mild infections such as surgical wound infections. Surgical complications that can predispose to infections include leaks, adhesions and obstructions, fistula formation, and urosepsis following urinary conduit formation. Once again, these complications can be readily identified, characterized, and in many cases, managed by utilizing imaging and interventional procedures.

Role of imaging in the cancer patient infections

Imaging plays an essential role in evaluating infections in cancer patients, and radiologists can facilitate early detection and prevention of infection in this fragile patient population, thus truly adding value to patient care with potential to improve outcomes. Abdominal radiographs have been largely replaced by cross-sectional imaging [28], although due to its portability, is performed for the evaluation and surveillance of bowel obstruction and pneumoperitoneum, as well as to evaluate implanted devices and catheters. Ultrasound (US) is another technique that can be utilized portably, which in the abdomen and pelvis is mainly utilized in the assessment of hepatobiliary, genitourinary, and gynecologic infections and related pathologies. Evaluation of the bowel can be sometimes difficult due to poor transmission of sound waves through the gas. Operator dependence, patient body habitus and limited cooperativeness can markedly affect the diagnostic accuracy of ultrasound. CT has become the modality of choice in the workup of patients with abdominal infections, as well as in patients with nonspecific presentations such as fever or sepsis. MRI is a useful modality particularly in hepatic or pancreaticobiliary imaging and can aid in the differentiation of metastases from infection-related pathology, assess biliary obstruction, and guide lesion characterization [29]. The main drawback of

MRI is that its acquisition is longer than CT and difficult to perform in clinically unstable patients due to longer breath-holds; in cancer patients additional limitations include pain, frequent movements, and inability to lay flat or to follow commands. Functional imaging with scintigraphy, namely with indium-111 white blood cell (WBC) scan, had been classically used for fever of unknown origin but now is largely replaced by anatomic imaging, although it is still currently one of the most useful imaging examinations for evaluating vascular graft infection [30]. Improved localization with SPECT/CT increases the accuracy of identifying infections. Compared to WBC scan, FDG-PET/CT is more widely available, less costly, provides higher resolution, and is easier to semiquantify; this has enabled FDG PET/CT to gain grounds in evaluating fever of unknown origin [31]. Additional applications for evaluating infection with functional imaging have been described but are not routinely used [32].

Gastrointestinal tract infections

The immunity of the gastrointestinal tract is influenced by a number of factors including gastric acidity, intestinal motility, normal bowel flora, mucosal integrity, and humoral and cell-mediated immunity [33]. When any of these protective mechanisms are disrupted, infection can ensue. The gastrointestinal tract is a common site of infection in cancer patients, with causes including *Clostridium difficile*-associated colitis, *cytomegalovirus* (CMV) colitis, neutropenic enterocolitis, appendicitis, and intestinal perforation with secondary peritonitis and possibly abscess formation [34]. Clinical manifestations include abdominal pain, fever, diarrhea, and less commonly intestinal hemorrhage. The diagnosis is based on a combination of history, symptoms, serology and laboratory tests, histopathology, and radiologic findings. Management is most often conservative; interventional procedures and surgery are reserved for complications such as perforation or bleeding.

Neutropenic enterocolitis

Neutropenic enterocolitis, also referred to as neutropenic colitis or typhlitis, is a potentially life-threatening complication of chemotherapy, seen more commonly in hematologic malignancies following cytotoxic chemotherapy, especially in patients with acute myeloid leukemia. Pre-existing bowel conditions, such as diverticulitis, tumor infiltration, and previous surgery, increase the risk of neutropenic enterocolitis following chemotherapy [35]. Isolated organisms in neutropenic enterocolitis include various bacterial and fungal pathogens, with frequent polymicrobial infections. Bacterial pathogens include gram-negative bacilli, gram-positive cocci, and anaerobes, whereas Candida species is the most

common of fungal etiologies [35]. Patients typically present with fever and right lower quadrant pain, as the cecum is the most commonly involved site, possibly due to its distensibility and relative hypovascularity [36]. Symptoms typically appear in severely neutropenic patients (cell count < 500 cells per mm^3) in the third week after chemotherapy (median 17 days), which is the time of maximal mucosal damage and most profound neutropenia [35].

The accurate diagnosis of neutropenic enterocolitis depends on the correlation of clinical and imaging features. CT is the modality of choice for diagnosis due to its lower rate of false negatives (15%) compared to ultrasound (23%) and radiography (48%) [37]. On CT, the hallmark of neutropenic colitis is predilection to the right colon (cecum and ascending colon, with possibility of involving the terminal ileum), with non-specific features including colonic wall thickening, edema predominately affecting the submucosal layer, mucosal hyperenhancement, and inflammatory changes in the pericolonic fat. Although CT findings may be non-specific, if the extent of thickening is relatively marked, in the correct clinical setting, the diagnosis can be strongly suggested. In cases of perforation, which is uncommon, pneumatosis, extraluminal gas, and pericolonic fluid may also be seen [38, 39] (Fig. 6). US remains a reasonable alternative to CT, particularly in pediatric patients; patients with a wall thickness of > 10 mm on US, have higher mortality rates and prolonged clinical symptomatology [40, 41]. Differential diagnosis includes pseudomembraneous colitis and cecal diverticulitis.

Clostridioides difficile colitis

C. difficile, previously *Clostridium difficile*, is a gram-positive anaerobe that colonizes the bowel after antibiotic administration alters the normal gut flora. It is the most common cause of healthcare-associated infections, and cancer patients are twice more likely than other inpatients to develop hospital-onset *C. difficile* infection [42]. Patients usually develop *C. difficile* colitis a few weeks after antibiotic administration, and 10–25% of patients develop recurrent *C. difficile* infection [43]. The spectrum of *C. difficile* infection can range from mild to fulminant colitis. Fulminant colitis is characterized by hypotension or shock, ileus, and/or megacolon [43]. Other severe complications include bowel perforation, septicemia, and death, with mortality rates of 5% from the colitis itself, and 15–25% from its complications [43]. On imaging and endoscopy, ulcerations in the mucosal lining can be seen in approximately 50% of cases and produce typical pseudomembranes. This finding is highly suggestive of *C. difficile* but not entirely specific, as pseudomembranous colitis can also be caused by inflammatory conditions and other infectious pathogens such as CMV and enterohemorrhagic *E. coli* [43]. Imaging features of *C. difficile* colitis may be non-specific, with wall thickening, submucosal edema, mucosal hyperenhancement, and pericolonic fat stranding noted. *C. difficile* infection usually involves the entire colon, and less commonly presents with segmental involvement, or with limited right-sided colitis in 30–40% of cases [38, 44]. Mucosal hyperenhancement may evolve to a lack of enhancement and mucosal sloughing. *C. difficile* produces one of the most severe forms of colitis due to transmural edema, with wall thickening up to 32 mm (average of 15 mm), which, along with mucosal hyperemia, results in the "accordion sign" (Fig. 7) [39]. "Accordion sign" refers to the appearance of the colonic wall due to the oral contrast entrapment between edematous haustral folds. Another characteristic finding is irregular mucosa with polypoid protrusions resulting in "wall nodularity," which is equivalent to "thumbprinting" that is seen in *C. difficile* infection on contrast enema and radiographs

Fig. 6 *E. coli* neutropenic enterocolitis in a 65-year-old woman with history of large B cell lymphoma, who presented with sepsis after chemotherapy treatment. **a** Axial and **b** reconstructed coronal intravenous contrast-enhanced CT images demonstrate marked thickening of the stomach (long arrow), duodenum and small bowel (short arrows), and large bowel (arrowheads) wall with associated mucosal hyperenhancement and low attenuation submucosa, compatible with edema. Small volume ascites is also noted (asterisks)
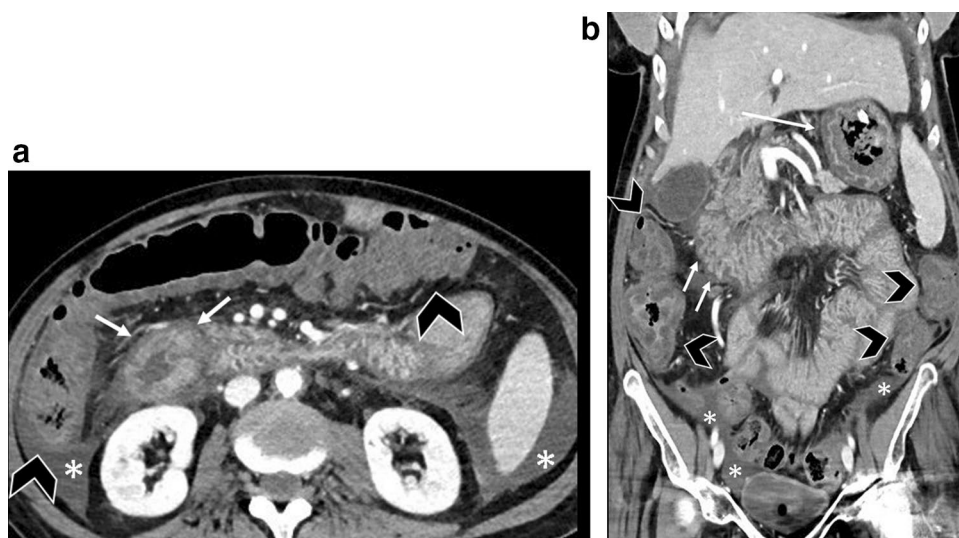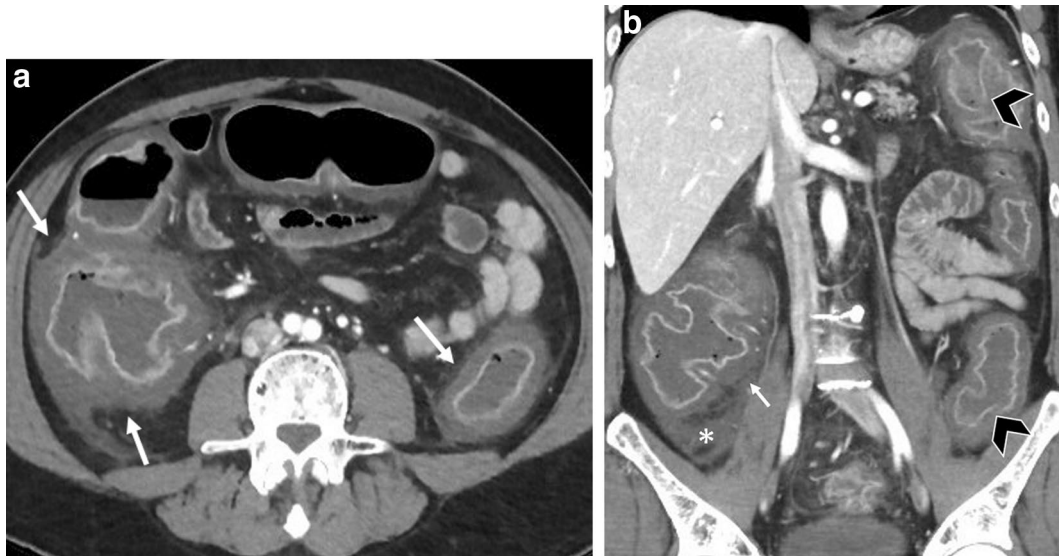
Fig. 7 C. *difficile* induced pseudomembranous colitis in a 62-year-old man with large B cell lymphoma, who presented with fever and severe diarrhea. **a** Axial and **b** reconstructed coronal intravenous contrast-enhanced CT images show marked pancolitis, manifested by marked wall thickening of the entire colon with avid mucosal hyperenhancement and pericolonic fat stranding (short arrows). Note sloughing of the mucosa resulting in the pseudomembranous appearance (arrowheads). There is trace reactive ascites (asterisk)

[39]. Major complications of *C. difficile* colitis include ileus, toxic megacolon, colonic perforation, and septic shock [45]. Toxic megacolon should be radiologically suspected when colonic diameter exceeds 6 cm in the setting of colitis [46]. Management is usually medical; however, in complicated cases, surgery may be required.

Viral and fungal enterocolitides

Several additional gastrointestinal infections encountered in cancer patients are less common but pose a high degree of morbidity and are important for radiologists to recognize. The mortality rate of CMV colitis in cancer patients reaches 42%, with the colon, stomach, and esophagus being the most common sites of CMV [47]. Imaging features of gastrointestinal CMV infection are also non-specific, and include wall thickening, ascites, and lymphadenopathy (Fig. 8). Nonetheless, when CMV infection becomes angioinvasive and produces ulcers and other ischemic or perforation-related changes, the findings on imaging can be suggestive. It is important to keep in mind that many patients frequently have overlapping processes especially in patients after stem cell transplant who may present with both CMV and GVHD.

Norovirus is the most common cause of epidemic gastroenteritis worldwide and the most common GI tract pathogen to cause hospital Emergency Department visits in the USA [48]. Norovirus affects 17–18% of immunosuppressed patients, and treatment consists of supportive measures [48]. CT findings in norovirus infection include small bowel wall thickening and fluid-filled small bowel, whereas the stomach
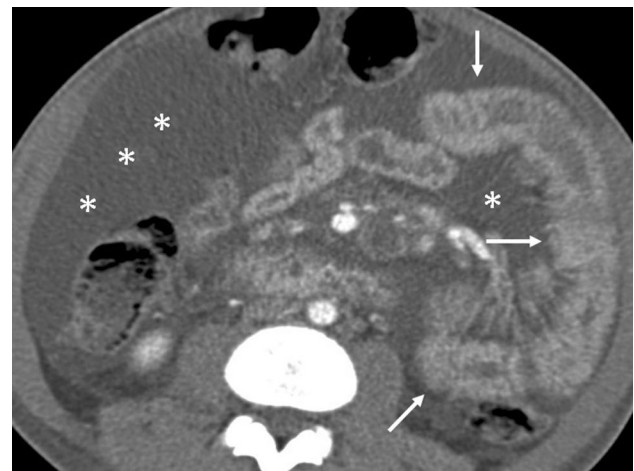
Fig. 8 Cytomegalovirus (CMV) enterocolitis in a 34-year-old man with acute myelogenous leukemia, who presented with abdominal pain 18 days after stem cell transplant. Axial intravenous contrast-enhanced CT image in the late arterial phase demonstrates wall thickening and diffuse hyperenhancement of small bowel loops (arrows) with large-volume ascites (asterisks). Note that the bowel loops are not distended

and colon are typically spared [49]. The thickened small bowel wall seen in norovirus is typically low in attenuation, similar to other causes of enteritis, but a differentiating feature from neoplastic bowel infiltration and mural hemorrhage, both of which demonstrate hyperdense small bowel wall [50].

Gastrointestinal fungal infections are becoming increasingly common as the population of immunocompromised patients continues to grow [51], which led to the introduction of azole prophylaxis (e.g. fluconazole and itraconazole) particularly for patients with acute leukemia and subsequent decrease in incidence [52]. Although Aspergillus and Candida species comprise the majority of fungal pathogens, Mucorales trends show an increasing incidence, especially in cancer patients and diabetics [51]. In immunocompromised patients with abdominal aspergillosis infection, CT demonstrates the involvement of solid organs, gastrointestinal tract, lymph nodes, peritoneum, and/or abdominal wall, with imaging features including hepatic and splenic microabscesses, concentric bowel wall thickening, vascular thrombosis with secondary infarcts, diffuse peritoneal thickening, and nodular retroperitoneal infiltration (Fig. 9) [53]. Mucorales are histologically similar to Aspergillus, and both species are angioinvasive [51]. Gastrointestinal mucormycosis is uncommon but has a high mortality of 40–50% and causes large ulcers with irregular edges within the stomach and the colon (Fig. 10) [51]. Otherwise, gastrointestinal fungal infections produce non-specific imaging findings such as bowel wall thickening and inflammation, and there is potential to spread to the peritoneum resulting in secondary peritonitis [54]. In severe disseminated disease, *Candida* may deposit on the serosal surface of the GI tract. On pathology, candida enterocolitis causes ulcers, sloughed mucosa, and infarcts [55]. Even if a specific diagnosis cannot be reached, imaging is useful to monitor response to treatment and detect complications.
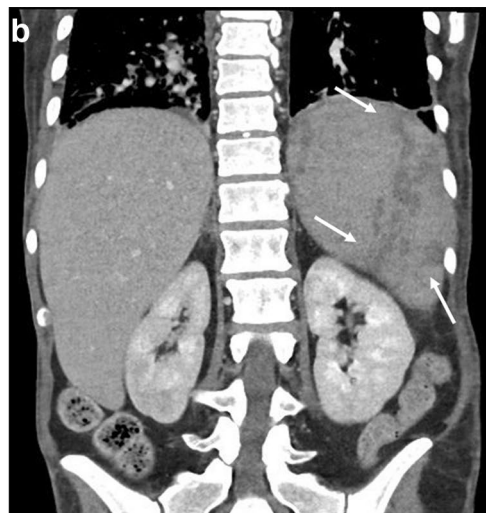

Fig. 10 Gastrointestinal mucormycosis in a 58-year-old woman with myelodysplastic syndrome, neutropenia, and melena. Reconstructed coronal intravenous contrast-enhanced CT image demonstrates focal severe wall thickening and dilatation of the hepatic flexure of the colon with irregular mucosal contour (arrows) consistent with typical mucor ulcers. Most of the colon demonstrates normal radiologic appearance (arrowheads)

Perianal and perirectal infections

Perirectal infections in cancer patients are very common, and normal imaging findings do not exclude this diagnosis,

Fig. 9 Invasive aspergillosis in a 55-year-old woman with acute myelogeneous leukemia. **a** Axial contrast enhanced CT image demonstrates peripheral wedge-shaped consolidation in the right lung base (arrowheads). **b** Reconstructed coronal CT shows multiple wedge-shaped and serpiginous areas of hypoattenuation in the spleen (arrows) which were consistent with infarcts from aspergillosis on autopsy

especially in neutropenic patients who have limited ability to mount an inflammatory response [56]. Radiation is another major risk factor for perirectal infections. Definitive or neoadjuvant pelvic radiation is a mainstay of treatment for prostate, anorectal, and gynecologic cancers. Acute proctitis usually develops within the first 6 months following radiation, mostly resulting from mucosal inflammation, and affects 20% of patients receiving pelvic radiation [57]. Chronic radiation proctitis affects 5% of patients after radiotherapy and is primarily caused by microvascular insufficiency from obliterative endarteritis. This leads to intestinal ischemia, resulting in transmural fibrosis and possibly strictures, ulcerations, fistulas, and perforation [57]. The leading clue in diagnosis is a history of radiation therapy; additional symptoms at presentation include diarrhea, tenesmus, and hematochezia [57]. On imaging, CT demonstrates mural stratification and wall thickening that is usually confined to the radiation port (Fig. 11). Fistulas are relatively common after pelvic radiation, and fluoroscopy is often needed to better characterize fistulous tracts; management can be challenging with implications for the quality of life. MRI has high soft-tissue resolution and can delineate the extent and degree of sphincter involvement of fistulous tracts as well as any associated small abscesses in the perianal region. MRI is also superior to CT in differentiating tumor recurrence from an infectious process [58].

Hepatobiliary infections

Hepatobiliary infection refers to infectious cholangitis, acute and chronic viral hepatitis, and gastrointestinal or systemic infections that manifest in the liver (due to its portal circulation and role in defense against microorganisms). Bacterial, mycobacterial, parasitic, and fungal infections can affect the hepatobiliary system [59]. Hepatosplenic candidiasis is the most common form of chronic disseminated candidiasis, and it is almost exclusively encountered in patients with hematologic malignancies after chemotherapy (Fig. 12) [60]. Candida presents with multifocal microabscesses that develop predominately in the periphery of the liver, spleen, and, sometimes, renal cortex. CT and MR imaging can demonstrate microabscesses in approximately 90% of patients, whereas US reveals them in 70%–75% of patients [61]. On US, the microabscesses can be hypoechoic or demonstrate a "bull's eye" appearance, which describes a peripheral hypoechoic fibrotic ring and central hyperechoic inflammation [62]. MRI and CT demonstrate innumerable tiny microabscesses, typically subcentimeter in size that are hypoattenuating on contrast-enhanced CT. MRI findings depend on the phase of infection as in the acute phase the microabscesses are hypointense on T1-weighted and hyperintense on T2-weighted images, in the subacute phase the microabscesses develop a hypointense peripheral ring on T1- and T2-weighted images with a central hyperintense signal on T1-weighted images, and in the chronic phase
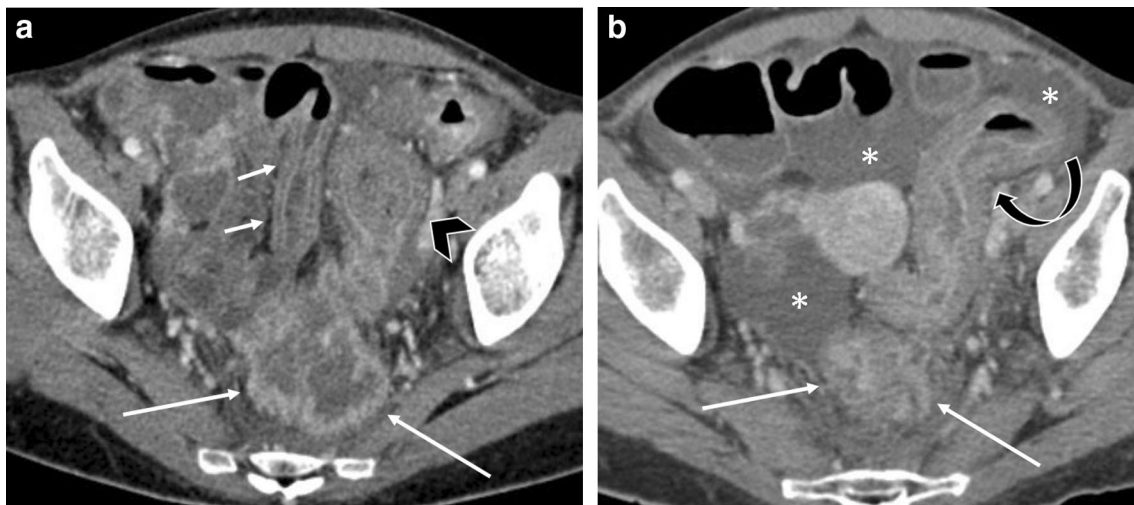
Fig. 11 Acute radiation enterocolitis in a 29-year-old woman with rectal cancer following neoadjuvant chemoradiation, who presented with fever and diarrhea 5 days before the completion of 50 Gy of radiation. **a**, **b** Axial intravenous contrast-enhanced CT images obtained immediately after treatment (**a**) and 9 days later (**b**). Initial CT shows mild wall thickening and mucosal hyperenhancement of small (short arrows in **a**) and large (arrowheads in **a**) bowel loops, with discontinuous involvement confined to the radiation port. **b** Repeat CT demonstrates increased wall thickening and diffuse mucosal enhancement of the sigmoid colon (curved arrow), with development of large-volume reactive ascites (asterisks). Worsening of bowel wall inflammation implies superinfection of the radiation-induced enterocolitis
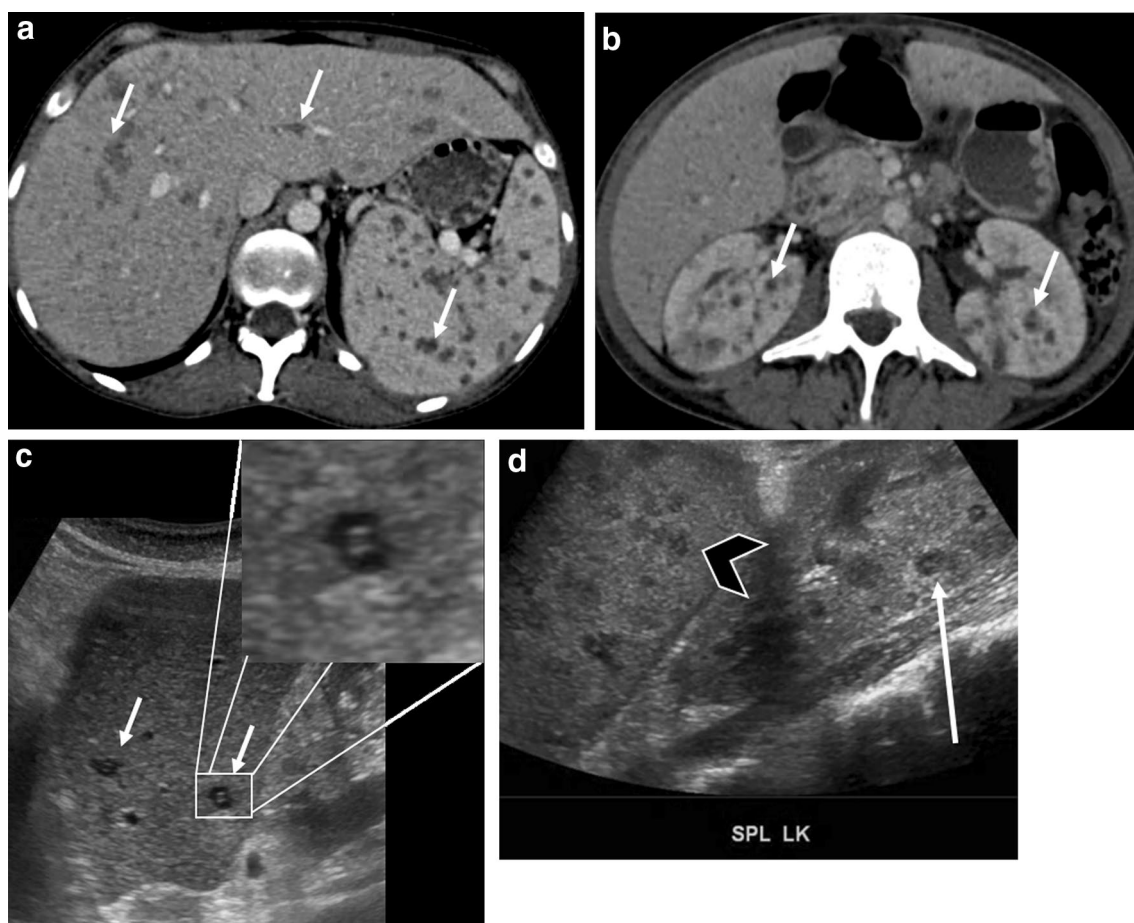
Fig. 12 Hepatosplenic and renal candidiasis in a 26-year-old man with acute myelogenous leukemia and severe neutropenia. **a**, **b** Axial intravenous contrast-enhanced CT images show innumerable hypoattenuating subcentimeter abscesses in the liver, spleen, and kidneys (arrows in **a, b**). **c**, **d** Gray scale ultrasound images obtained in trans-verse (**c**) and sagittal (**d**) planes demonstrate the typical "bull's eye" appearance of the lesions with a hypoechoic ring and an echogenic center in the liver (short arrow in **c**), spleen (arrowhead in **d**), and left kidney (long arrow in **d**). Inlet of the magnified lesion demonstrates "bull's eye" appearance

they become globally hypointense on both sequences and may regress [63]. Candidiasis micrabscesses can become calcified when treated. Hepatic metastases, lymphomatous foci, and sarcoidosis are the top differential choices for hepatosplenic candidiasis. Hepatic metastases usually are larger, less numerous, with splenic involvement only seen in the latest stages of cancer. Lymphomatous foci in the liver are also larger than lesions seen in candidiasis; however, the spleen is commonly involved.

Cholangitis

Acute, or ascending, cholangitis refers to a biliary infection in the setting of biliary stasis caused by an obstructing stone, stricture, or tumor. Indwelling biliary stents and choledochojejunostomy or hepaticojejunostomy also predispose to cholangitis due to easier retrograde translation of enteric bacteria. Acute cholangitis, in 10–30% of cases,

can be the initial presentation of an obstructive tumor of the gallbladder, bile ducts, pancreas, or duodenum [64]. The classic triad described by Charcot in 1877 consists of abdominal pain, fever, and jaundice, and is seen in around two-thirds of patients [65]. Antibiotics and endoscopic cholangiopancreatography (ERCP) with decompression (either by stone removal or the placement of stents to bypass any obstructing pathology) are the mainstay of treatment. Percutaneous biliary drainage remains an option if ERCP is not feasible or unsuccessful.

Acute suppurative cholangitis refers to cholangitis with the presence of pus in the biliary tract and is more often seen in elderly patients > 70 years of age, smokers, and in patients with impacted biliary stones [66]. It can lead to increased intraluminal pressure and predisposes to biliary sepsis. Acute cholangitis can also be procedure-related and is reported in 18% of patients after transhepatic percutaneous biliary drainage [67]. Bacterial pathogens in biliary

infections arise from the GI tract, and the most common bacterial organisms isolated in infected bile without prior instrumentation are *E. coli* (31%), *Klebsiella pneumoniae* (17%), *Enterococcus faecalis* (17%), and Streptococcus species (17%). In patients with plastic biliary stents, Enterococcus, and polymicrobial infections are most encountered [67].

Cross-sectional imaging of cholangitis demonstrates central, diffuse, or segmental biliary dilation, with smooth symmetric and diffuse bile duct wall thickening that is most pronounced in the central ducts. US is the best initial imaging test to assess for the presence of biliary dilatation/obstruction. In suspected cases of infectious cholangitis contrast-enhanced CT and MRI may show early, and intense enhancement of the thickened bile duct walls and the liver parenchyma may demonstrate wedge-shaped peripheral patchy peribiliary enhancement, findings that are most pronounced in the arterial phase (Fig. 13). US and MR cholangiopancreatography (MRCP) can determine the presence of tumors or stones in the central bile ducts. Pus may be seen on CT as hyperdense material within the distended ducts. Inspissated bile or sludge may also be hyperdense on CT; MRI, especially DWI, can be more specific for identifying purulent material, as it demonstrates restricted diffusion with very low signal intensity on ADC map but no internal enhancement. Cholangitis can be complicated by bacteremia and sepsis, hepatic abscesses, portal vein thrombosis, and bile peritonitis [67].

Liver abscesses

A liver abscess can develop from biliary, hematogenous, or contiguous spread. Bacterial, or pyogenic, hepatic abscess is the most common form in the developed world and carries a mortality rate of around 15% [29]. In patients with cancer, mortality from liver abscesses is twice as high, and in patients with hepatobiliary malignancies, 6-month mortality rates from uncontrolled sepsis and/or from progressive cancer is even higher at 72% [68]. Neoplasia and biliary tract disease are the main causes of liver abscesses [69], which may develop after chemotherapy, following interventions, including liver biopsy, intra-arterial and percutaneous embolization procedures (Fig. 14), and after surgical procedures such as partial liver resections, hepaticojejunostomy, pancreaticoduodenectomy, and other bowel surgeries [29, 68, 69]. In addition to fever and chills, patients present with nausea, vomiting, and jaundice [68]. In patients with primary hepatobiliary malignancies, hepatic abscesses are more likely to communicate with the biliary tract, and pathogens are commonly *E. coli* and *K. pneumonia*. Patients with other solid malignancies have more diverse agents and polymicrobial infections [68]. Fungal hepatic abscesses are less common, and they tend to develop in patients with hematologic malignancies after chemotherapy [69].

The diagnosis of hepatic abscess depends mainly on imaging; and CT is the modality of choice in the acute and emergent setting. MRI can provide additional details regarding the communication of abscesses with the biliary
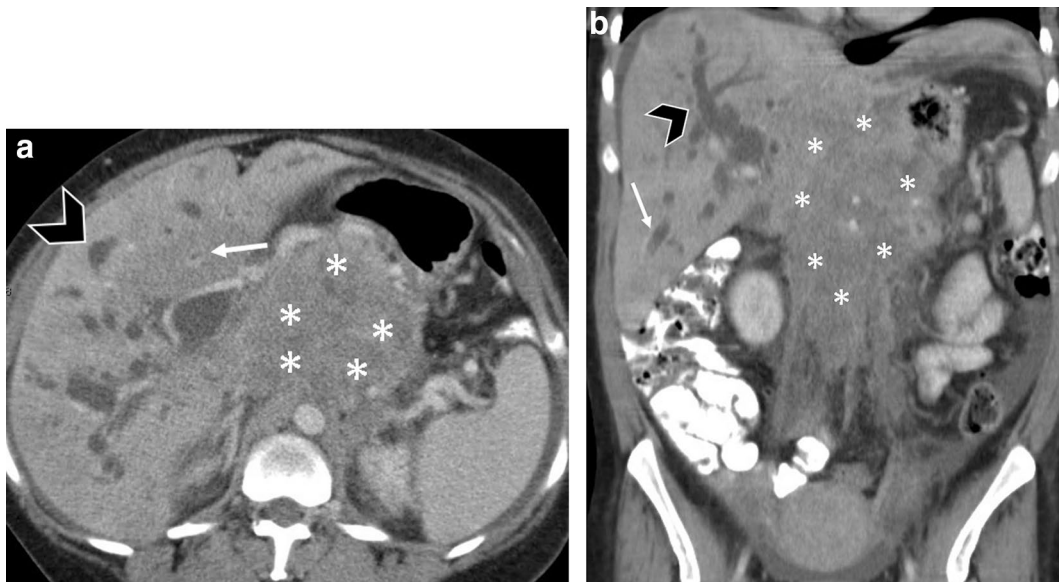
Fig. 13 Acute cholangitis and biliary obstruction in a 48-year-old woman with metastatic cervical cancer. **a** Axial and **b** reconstructed coronal intravenous contrast-enhanced CT images demonstrate confluent retroperitoneal lymphadenopathy (asterisks) causing mass effect and obstruction of the common hepatic duct resulting in intrahepatic biliary ductal dilation (arrowheads). There is circumferential enhancement of bile duct walls (arrows) in segmental distribution which is a feature of cholangitis
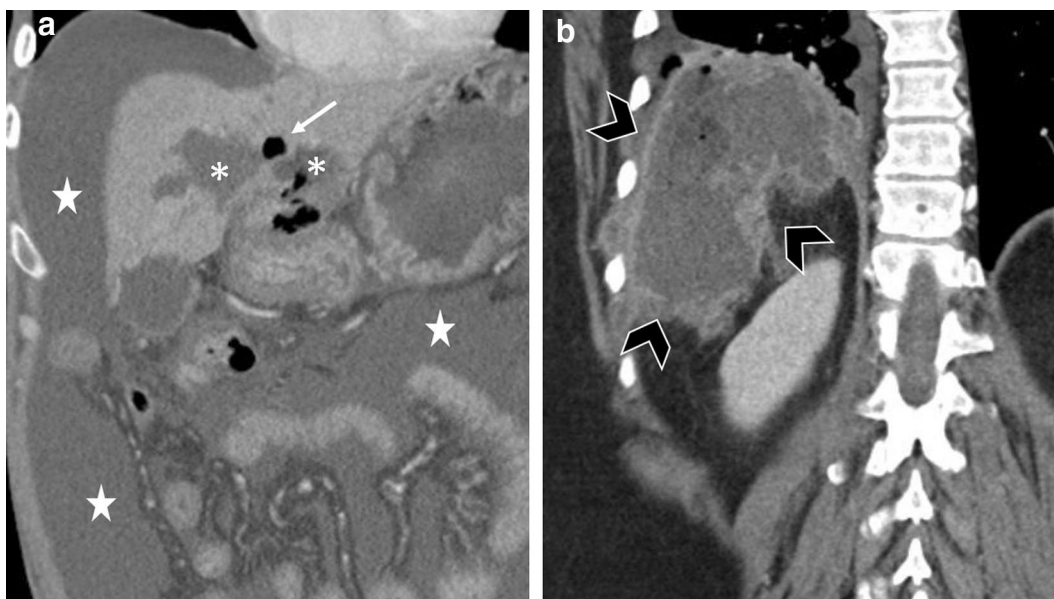
Fig. 14 Liver abscesses developed as a complication of post intra-abdominal procedures in two different patients. **a** 66-year-old man with metastatic pancreatic cancer and liver abscesses after biliary drainage, who presented with fever and abdominal pain 1 week after biliary drain placement. Reconstructed coronal intravenous contrast-enhanced CT image shows hypodense liver collections (asterisks) some of which contain gas (arrow) consistent with hepatic abscesses. Noted is large-volume ascites (stars). **b** 67-year-old man with metastatic pancreatic adenocarcinoma and liver abscess who presented with fever and flank pain 2 months after microwave ablation for a solitary hepatic metastasis. Reconstructed coronal contrast enhanced CT image demonstrates a large hepatic abscess under the right hemidiaphragm containing fluid and gas pockets with irregular enhancing rim (arrowheads) that yielded purulent fluid upon aspiration

tract (using MRCP or hepatobiliary contrast agents) and in differentiating hepatic abscesses from necrotic metastases. Abscesses demonstrate a rather thin and homogeneous wall, and greater restricted diffusion with lower ADC than metastatic deposits, with more rapid temporal evolution over days and weeks [70]. In abscesses, the restricted diffusion correlate with areas of high T2 signal, and occurs more centrally in the non-enhancing portion, while in necrotic tumors the restricted diffusion occurs in the more solid areas that have intermediate T2 signal intensity, and corresponds to the thick enhancing wall; in rare occasions, metastases can have superimposed infection. Percutaneous drainage is often required for treatment of liver abscesses that are larger than 6 cm, for abscesses with impending perforation, and when there is persistent fever after isolated medical treatment and aspiration [71].

Reactivation of viral infections

Reactivation of hepatitis B and hepatitis C viruses occurs in 20% of patients with known chronic hepatitis after receiving chemotherapy [72, 73] typically seen after targeted therapy, such as rituximab [74]. Clinical manifestations can range from asymptomatic or mild to life-threatening liver injury. Imaging findings in hepatitis are non-specific and reflect parenchymal and periportal edema, which can be seen on US

as a "starry sky" appearance due to increased echogenicity of the portal triads, and on MRI as diffusely heterogeneous signal intensity of the liver with hyperintense signal on T2 weighted images. Less commonly, this can manifest as areas of segmental edema (Fig. 15). After contrast administration on CT and MRI, the liver parenchyma demonstrates early and heterogeneous enhancement. Management typically consists of optimizing antiviral therapy and modifying the cancer treatment regimen. Chemotherapy has also been shown to cause reactivation of a variety of other viruses, including HSV, CMV, EBV, HHV6, and HHV8 [75].

Genitourinary tract infections

A wide variety of factors in cancer patients increase the risk of genitourinary infections, including obstructive uropathy from solid tumors, ureteral reflux from loss of the ureter-ovesical junction, indwelling catheters and stents, and pelvic radiation.

Obstructive uropathy

Urinary tract obstruction can occur at the level of the ureter, bladder, or urethra, causing urinary stasis which is a major risk for bacterial colonization and infection [76]. In
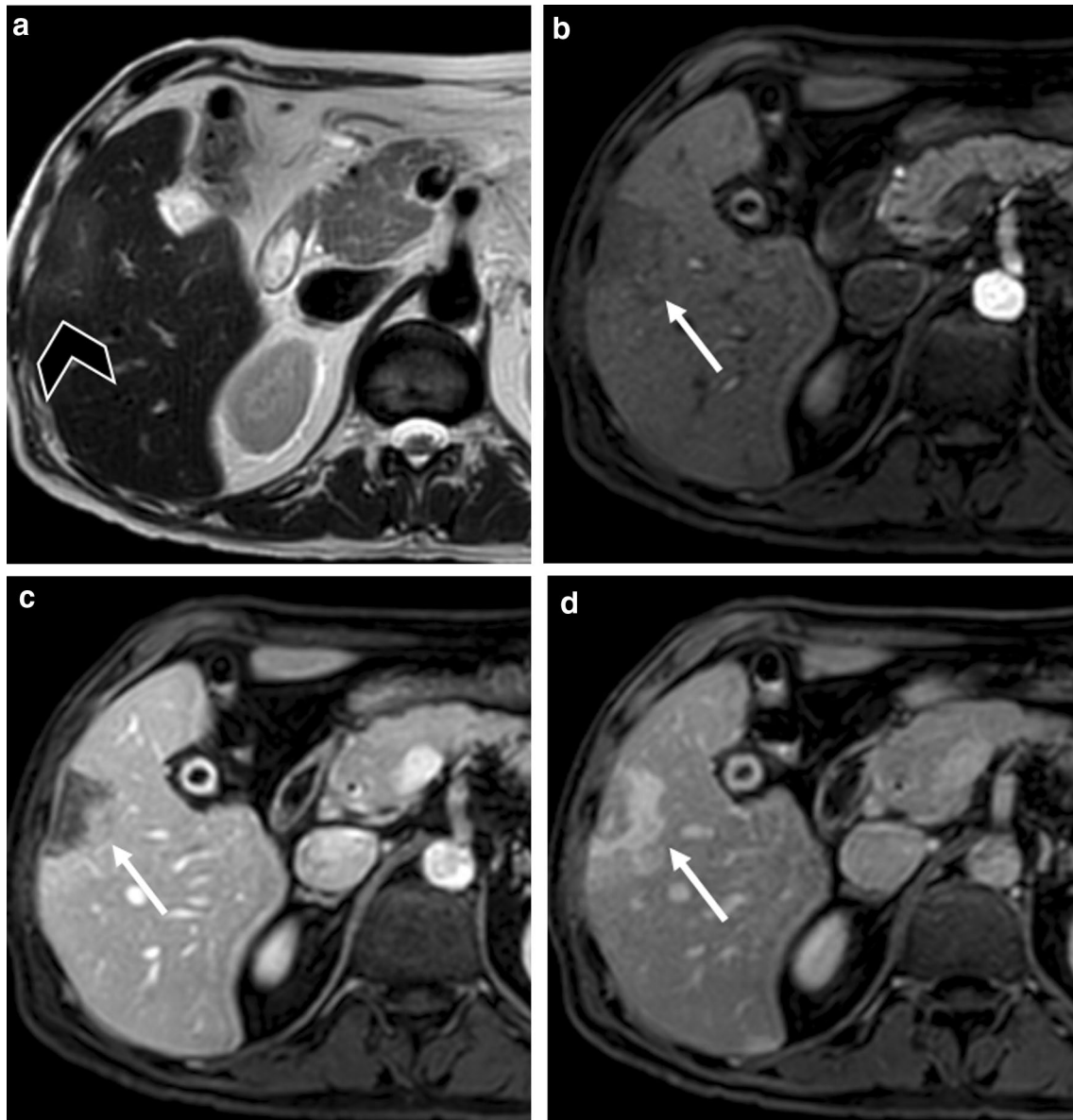
Fig. 15 Acute adenovirus hepatitis in a 49-year-old man with diffuse large B cell lymphoma and bone marrow transplant, who presented with high-grade fever. **a** MRI abdomen T2 weighted image obtained in the axial plane demonstrates subcapsular wedge-shaped area of faintly hyperintense signal in segment 5 (arrows). **b–d** MRI T1 weighted images in the axial plane obtained in arterial (**b**) equilibrium (**c**) and 5-min delayed (**d**) phases demonstrate no arterial enhancement (arrow in **b**), and progressive equlibrium and delayed fill-in (arrows in **a**, **b**), suggestive of an inflammatory or infectious process. Multiple similar subcapsular areas were also present (not shown). There was no mass effect and no vascular displacement. Liver biopsy showed inflammatory cells with no malignancy and adenovirus PCR was 2,478,000,000 copies/ml

addition to stasis, urinary obstruction affects renal function and decreases the amount of antibiotics excreted in the urine.

Ureteral obstruction can be caused by retroperitoneal adenopathy, pelvic malignancies (Fig. 16), primary ureteral malignancies, or radiation-induced stricture [3, 77]. US and CT are the main imaging modalities in evaluating obstructive uropathy and its complications. Hydroureteronephrosis and asymmetric nephrogram are the most direct signs of ureteral obstruction on CT. Ultrasound is safe and cost-efficient modality for the initial diagnosis of hydroureteronephrosis and serial follow up; and can also be used to evaluate for a change in renal pelvis dilation after voiding, and in estimating the post-residual bladder volume in cases of bladder outlet obstruction. CT urogram with either a split bolus technique or a 3-phase examination may also be used to delineate any filling defects in the collecting systems or ureters, which can include tumor, pus, stones, or fungal ball. MRI can isolate the level of
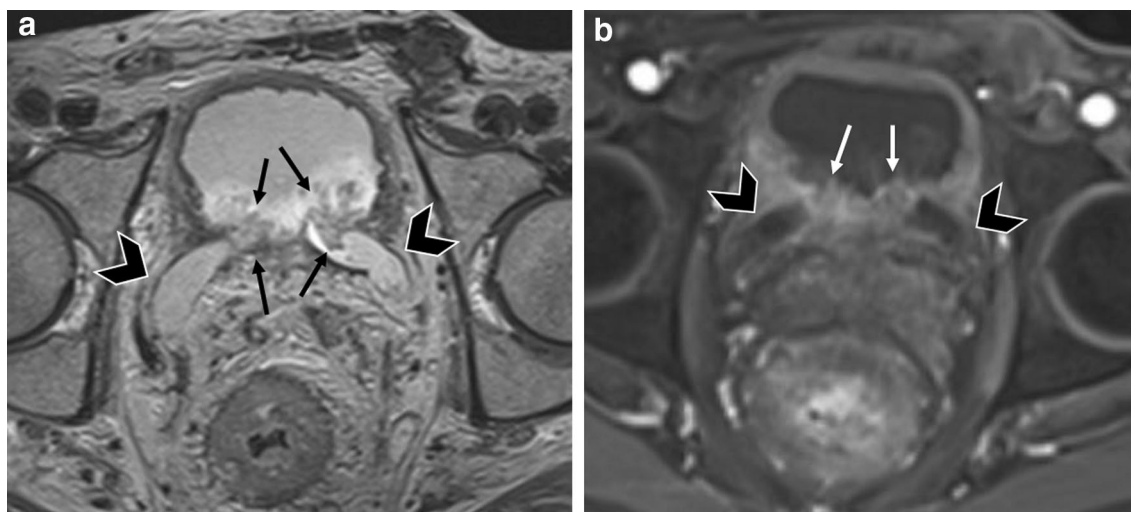
Fig. 16 Obstructive uropathy in a 69-year-old man with rectal adenocarcinoma and tumor local extension into both ureters, who presented with acute renal failure after completing chemoradiation. **a**, **b** MRI of the pelvis obtained in the axial T2-weighted (**a**) and contrast enhanced T1-weighted fat saturated arterial phase (**b**) images demonstrate irregular tumoral local infiltration (arrows) into the distal ureters and ureterovesical junctions bilaterally with bilateral ureteral dilation (arrowheads). Cystoscopy confirmed tumor replacing the entire base of bladder, including trigone and bilateral ureters, with pathology consistent with colorectal adenocarcinoma. Note the diffusely thickened and enhancing bladder wall typically seen with cystitis

obstruction and assess any soft tissue that may be causing extrinsic obstruction, but it is limited for small stones or urothelial abnormalities. Renal scintigraphy provides functional information about the urodynamic significance of obstruction and its effect on renal function. Management typically requires decompression with retrograde ureteral stents or percutaneous nephrostomy tubes.

Pyelonephritis, renal abscess, cystitis

Patients with urinary diversion following resection of a bladder tumor have a 20% risk of developing pyelonephritis [78]. Intestinal urinary pouches have chronic bacteriuria with *E. coli* as well as other gram-negative Enterobacteriaceae and gram-positive cocci [78]. Cancer patients with malignant obstruction, stricture, or leaks from radiation or from surgery require nephrostomy tube placement, which carries a similarly high risk of pyelonephritis, reported at 19% within the first 3 months after placement and caused by gram-positive (48%) and gram-negative (40%) organisms, and less often by Candida species (12%) [25]. *C. albicans* is the most common fungal organism to cause urinary tract infections, and imaging may demonstrate renal microabscesses similar to chronic disseminated candidiasis, or less commonly larger abscesses, renal infarcts, or fungal balls [54]. Neutropenia is an independent risk factor for the development of pyelonephritis in cancer patients with indwelling nephrostomy tubes, with an odds ratio of 3.2 [25]. In addition to antibiotics, nephrostomy tube infection treatment may require

catheter removal or exchange; however, recurrent infection remains a challenge [25].

Renal abscess typically develops in the setting of pyelonephritis (Fig. 17). Less likely, it can occur as a result of a contiguous infection or from the hematologic spread in the setting of bacteremia or fungemia. Antibiotics are the mainstay of treatment, and aspiration or drainage may be required depending on the abscess size and temporal evolution of symptoms, and is usually considered when the abscess size exceeds 5 cm [79]. Pyelonephritis should be suspected on US in cases of striated heterogeneous appearance of the renal cortex on gray-scale imaging associated with decreased perfusion on color/power Doppler. Similarly, contrast-enhanced CT may show wedge-shaped or rounded areas of streaky enhancement in the renal cortex, and provides a more comprehensive evaluation of the retroperitoneum. MRI, especially DWI, is very sensitive in depicting renal abscess and pyelonephritis. Renal abscess can be diagnosed when a new complex or cystic area is seen in the renal cortex with or without associated inflammatory changes in the perirenal fat.

Bladder infection can be a complication of an indwelling Foley catheter or suprapubic catheter. Imaging hallmark of cystitis includes diffuse thickening of the urinary bladder wall with or without inflammatory changes in the perivesical fat.

Fig. 17 Renal abscesses in a 32-year-old woman with cervical cancer who underwent hysterectomy and radiation treatment which were complicated by bowel and renal injury resulting in transverse colon conduit and loop ileostomy. Patient had persistent fever, weakness, and back pain and had two CT abdomen and pelvis performed. **a, b** Coronal reconstructed intravenous contrast-enhanced CT images obtained initially post therapy (**a**) and later post antibiotic treatment (**b**). Initial CT image demonstrates numerous small abscesses (solid arrows in **a**) in the right kidney. None of the collections were large enough for drain placement so the patient was placed on antibiotics. The patient continued to have persistent fevers and developed back pain. Follow-up CT image (**b**) demonstrates interval coalescence of previously seen innumerable abscesses into one large multiloculated renal abscess (asterisks). The patient improved after surgical drain placement (not shown). Note mild intrarenal collecting system fullness in both kidneys (dashed arrows in **a, b**)

Prostatic infections

Cancer patients can develop prostatic infections after procedures such as prostate biopsy, urethral or suprapubic catheter placement, brachytherapy and cryotherapy, and after intravesical BCG for bladder cancer [80]. Chemotherapy and bacteremia are additional systemic factors in cancer patients that increase the risk of developing prostatic abscess [80]. Presenting symptoms are dysuria, urgency, frequency, sensation of incomplete voiding, and suprapubic or perineal pain. In 60–80% of cases, acute bacterial prostatitis and prostatic abscess are caused by gram-negative bacteria, with *E. coli* being the most common pathogen [80]. Imaging features are non-specific in the setting of prostatitis, but the gland is usually enlarged and edematous. A prostatic abscess can be detected on transrectal ultrasound of the prostate as well as on CT, but MRI provides better characterization; and both CT and MRI can demonstrate a unilocular or multilocular rim enhancing collection, more commonly in the transition zone or central zone of the prostate (Fig. 18) [80].

Peritoneal, retroperitoneal, and abdominal wall infections

Intra-abdominal and abdominal wall infections can be overlooked in oncologic patients, but prompt recognition and management play an important role in prognosis. Malignancy is an independent factor for predicting outcome of peritonitis [81]. In addition to systemic patient factors, solid

Fig. 18 Prostate abscess in a 68-year-old man with small cell lung cancer, who presented after chemotherapy with urinary tract infection and urinary retention requiring an indwelling Foley catheter. Axial intravenous contrast-enhanced CT image demonstrates a fluid collection (asterisk) with enhancing wall (arrows) consistent with an abscess occupying almost the entire right peripheral zone of the prostate. A Foley catheter is noted within the prostatic urethra

tumors and treatments, including radiation therapy, paracentesis, surgery, and medical devices, increase the risk of

cancer patients for developing intra-abdominal and abdominal wall infections.

Peritoneal infections

Peritonitis can be infectious or non-infectious. Sterile peritonitis can occur after the introduction of a chemical irritant such as bile or blood. The main concern with peritonitis is a systemic inflammatory response that ensue with sepsis with a mortality rate of 30% [81].

Peritonitis can result from bowel obstruction and perforation, anastomotic dehiscence, or gastric perforation [80]. Cancer contributes to 10–16% of cases of gastric perforation, and spontaneous perforation carries a very high mortality up to 82% [81]. Ischemia, infectious enterocolitis, ulcers, and radiation enteropathy may also result in bowel perforation. Additional iatrogenic causes of peritonitis include surgery, indwelling peritoneal dialysis catheters, non-tunneled catheters and shunts. Bevacizumab has been reported to cause spontaneous bowel perforation without tumor involvement of the bowel [82]. Patients present with generalized abdominal pain, tenderness, guarding, and fever. Pathogens in peritonitis depend upon the cause, as pathogens in the upper gastrointestinal tract differ than those of the lower. US can be used to evaluate for ascites and collections, and to guide aspiration, but CT is the modality of choice in imaging peritonitis to identify a source and look for intra-abdominal abscesses. Typical imaging features are ascites, peritoneal enhancement and thickening, which is typically smooth with infectious etiology, but could less commonly be nodular or irregular, a feature more indicative of carcinomatosis (Figs. 19, 20).

Neutropenia, immunosuppressant, and antibiotic prophylaxis have led to an increasing prevalence of fungal peritonitis by Candida species [81]. Mycobacterial peritonitis is primarily seen in endemic areas, but has an increasing prevalence in the developed world due to immunosuppressive therapy and immigration; both *M. tuberculosis* and mycobacterium avium complex infections can mimic peritoneal carcinomatosis at presentation due to irregular peritoneal thickening and nodularity [83, 84]. Additional CT imaging features include thickened mesenteric root fat planes and lymphadenopathy characterized by ring enhancement and central hypoattenuation [84]. On ultrasound, multiple septations are usually present within complex ascites.
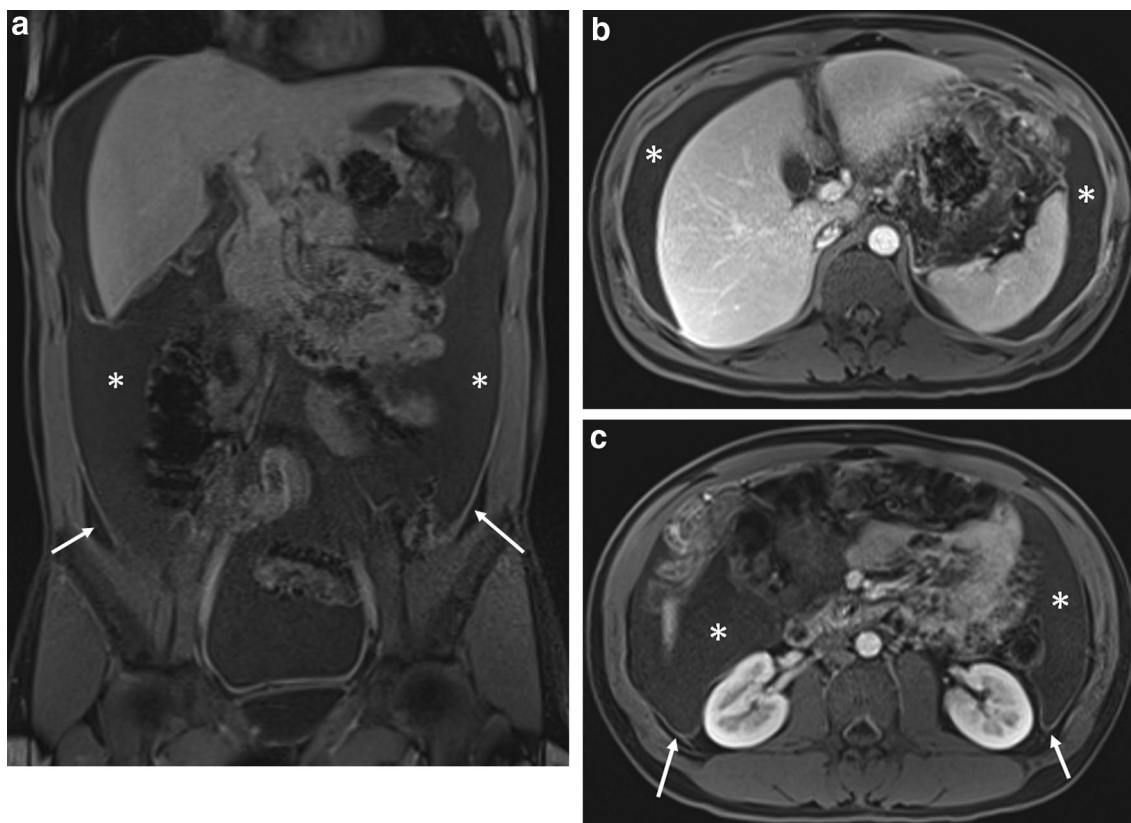
Fig. 19 Peritoniteal coccidiomycosis in a 45-year-old man with Non-Hodgkin's lymphoma on rituximab. **a** Coronal and **b, c** axial post-contrast T1-weighted MRI demonstrate marked peritoneal thickening and enhancement (arrows) with large-volume ascites (asterisks). Findings are consistent with infectious fungal peritonitis from coccidioides

Fig. 20 Infectious peritonitis in a 64-year-old woman with gastric cancer and peritoneal carcinomatosis. The patient presented with severe abdominal pain and clinical signs of peritonitis. **a, b** Axial intravenous contrast-enhanced CT abdomen and pelvis images demonstrate hyperdense peritoneal folds (solid arrows) and ascites (asterisks). Note extensive peritoneal carcinomatosis (dashed arrows in **b**)

Peritoneal devices

Patients with advanced abdominal malignancies often develop refractory ascites. Indwelling peritoneal catheters can be an alternative to repeated paracentesis and have an infection rate of 5.4% in the setting of malignant ascites, with a significantly increased infection risk when left for more than 12 weeks [85]. Simple fluid collections or ill-defined fluid around devices can be due to seromas and post-surgical changes; however, the development of an enhancing wall or new gas pockets without recent intervention is concerning for abscess formation. Certain types of peritoneal drainage catheters can be tunneled in the subcutaneous tissues, decreasing the rate of leaks and infection [85]. Additional peritoneal devices include peritoneal infusion catheters, dialysis catheters, ventriculoperitoneal shunts, and surgical drains; any of those can be complicated by peritonitis from translocation of skin flora or from bowel perforation, albeit the latter is rare. Peritonitis is also an uncommon risk following percutaneous gastrostomy tube placement. Management consists of antibiotics and removal of the causative device, with surgery in cases of frank perforation.

Intra-abdominal abscesses

An abscess, by definition, has an inflammatory wall and contains a viscous interior [86]. Several classification systems exist for intra-abdominal abscesses. By location, non-visceral abscesses are either peritoneal or retroperitoneal. Peritoneal abscesses are generally a complication of peritonitis
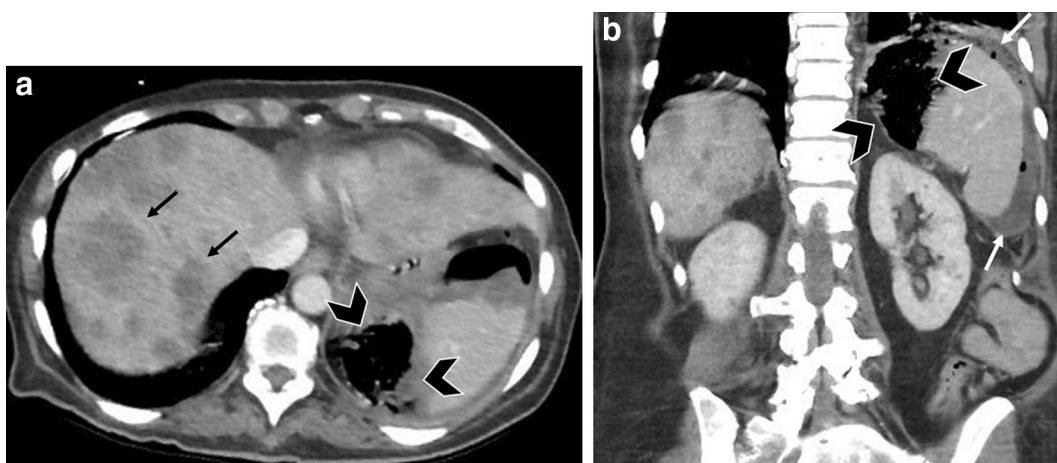
Fig. 21 Splenic clostridia abscess in a 71-year-old woman with advanced pancreatic cancer. **a** Axial and **b** reconstructed coronal intravenous contrast-enhanced CT images demonstrate a heterogeneous predominantly gas containing complex collection in the medial spleen (arrowheads), in addition to perisplenic rim-enhancing collection containing fluid and a few gas pockets (white arrows). Culture was positive for polymicrobial clostridioides. Noted are numerous liver metastases (black arrows)
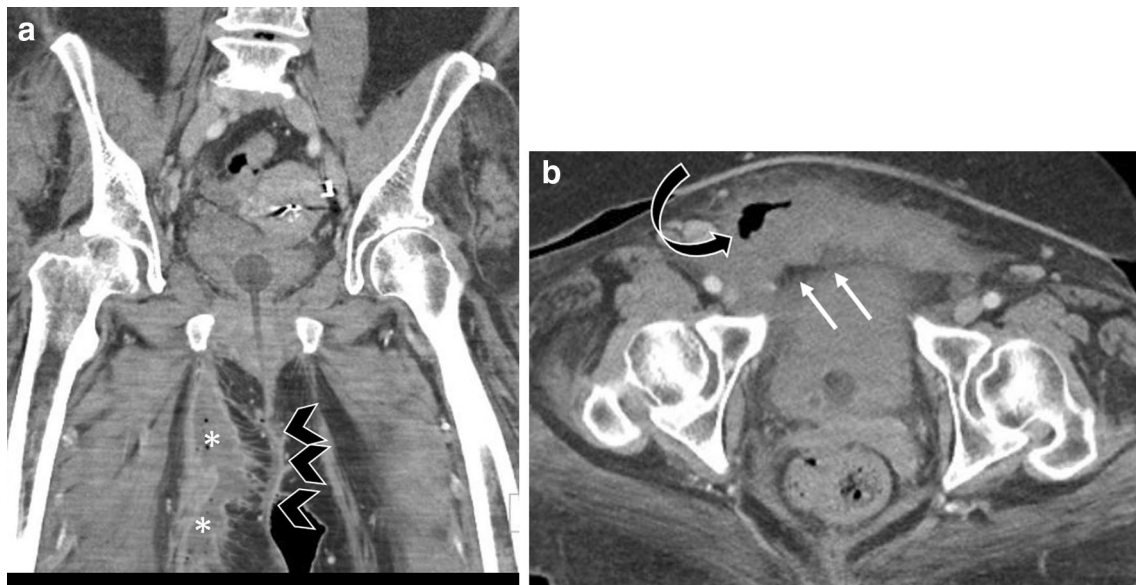
Fig. 22 Soft-tissue infection complicating radiation therapy in a 65-year-old woman with endometrial carcinosarcoma and type 2 diabetes mellitus. **a** Coronal reconstructed intravenous contrast-enhanced CT image of the pelvis demonstrates skin thickening and breakdown in the medial right thigh (arrowheads) associated with a large complex fluid collection with air within (asterisks) and hyperdense rim (arrow) in the soft tissues deep to the skin compatible with skin necrosis and soft tissue abscess. **b** Axial contrast enhanced CT pelvis image in the same patient demonstrates marked inflammatory and edematous changes in the right anterior pelvic wall (arrows), with central gas (curved arrow), highly consistent with necrotizing fasciitis. Patient underwent surgical debridement with slow wound healing which delayed her scheduled chemotherapy. Note Foley catheter within the decompressed bladder

and/or perforation. Retroperitoneal abscesses can be caused by hollow viscous perforation or by hematogenous, lymphatic, or local spread of infection. The vast majority of intra-abdominal abscesses are polymicrobial (Fig. 21) [86]. Presenting symptoms include fever and abdominal discomfort, with additional symptoms depending on the location of the abscess. For example, a perirectal abscess may cause diarrhea, and an abscess in contiguity with the bladder may cause urinary symptoms. Prolonged ileus should raise concern for an abscess in the post-operative setting [86]. Post-contrast CT and MR imaging demonstrate a rim enhancing fluid collection with surrounding inflammatory changes. Drainage is the mainstay of treatment, but antibiotics alone might be sufficient when the abscess is less than 3 cm in size [81]. Surgery is indicated when there is a need for source control by resection of the infected structure (example appendectomy) or ischemic bowel, debridement of necrotic tissue, or repair of traumatic perforations [81]. If left untreated, abscesses may extend to adjacent structures, erode into vessels (causing pseudoaneurysms, hemorrhage, and thrombosis), rupture, or less commonly fistulize, eventually leading to bacteremia and septic shock with high mortality rates [81].

Abdominal wall infections

Skin and soft-tissue infections (SSTI) in the abdominal wall can be very serious in immunocompromised patients, particularly those with vascular pathologies such as endarteritis obliterans (seen with radiation therapy) [87]. SSTIs include cutaneous infections in addition to deeper subcutaneous, muscular, and fascial infections such as cellulitis, necrotizing fasciitis, and pyomyositis. Deep SSTI infections can be caused by skin injury or skin disruption from surgery, catheter and line insertions, radiation treatment, and primary or metastatic tumors (Fig. 22). Cellulitis is a clinical diagnosis, but imaging features include skin thickening with subcutaneous fat stranding, edema, and inflammation. The presence of subcutaneous gas on non-contrast CT that spreads along fascial planes is usually worrisome for necrotizing fasciitis, requiring early and aggressive management with drainage and surgical debridement. Vesicocutaneous and enterocutaneous fistulas due to tumors, radiation therapy, or surgical complications can lead to SSTIs. Imaging with US, CT, or MRI can be helpful in delineating the predisposing factor, differentiating acute versus fibrotic fistula track as well as the number and relationships of tracts, and evaluating for any associated drainable abscesses.

Conclusion

Radiologists should be familiar with the risks of infection in oncologic patients and recognize the most common imaging manifestations of infections in patients with solid tumors and hematologic malignancies. Imaging plays an important role in diagnosis, management, and prognosis of infectious processes in the abdomen and pelvis in patients with oncologic conditions, including those affecting the gastrointestinal, hepatobiliary, and genitourinary systems, in addition to non-visceral and abdominal wall infections, and those associated with medical devices, radiation, and surgical procedures.

Compliance with ethical standards

Conflict of interest Authors have no relevant disclosures.

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