The most common complaints among patients with cancer who present to the emergency department (ED) are related to the gastrointestinal system, and 40% of these patients complain of abdominal pain. These presentations can stem from the underlying malignancy itself, the treatment directed toward the disease, or the full range of pathologies present in a healthy population. Moreover, immunosuppression may blunt many of the findings one expects in a healthy population of patients, thus rendering the clinical examination less reliable in patients with cancer. This article focuses specifically on patients with cancer who present with an acute abdomen, and discusses how a concurrent malignancy can shape the differential diagnosis in these cases.

To facilitate an organized approach to this article, the authors have considered these processes within three broad categories for the acute abdomen in patients with cancer: intestinal obstruction, intestinal perforation, or pathology unique to immunosuppression and malignancy.

THE IMPACT OF IMMUNOSUPPRESSION

Clinical findings and the spectrum of infectious processes are affected by the patient’s degree of immunosuppression. For simplicity, these can be classified as mild-to-moderate or severe forms of immunosuppression (Table 1). Patients in group I (mild-to-moderate) are not undergoing chemotherapy, though they have blunted symptoms due to their lack of an appropriate inflammatory response. Because of this, they often seek medical attention later in the course of their disease. Other than the direct impact of the malignancy itself, however, these patients have pathologies similar to those of a healthy population. Patients with profound immunosuppression associated with chemotherapy (group II) are more challenging. It is in this group of patients that classic examination findings of fever and abdominal tenderness may be replaced by vague signs or symptoms such as tachycardia, hypotension, and confusion. Concurrently, the differential diagnosis must be expanded to include a wide variety of atypical opportunistic infections.
MALIGNANT BOWEL OBSTRUCTION

In the United States, intestinal obstruction is a common cause of hospitalization, representing roughly 15% of all emergency admissions for abdominal pain, or 300,000 hospitalizations per year. This risk increases in patients with cancer—approximately 3% per year as a group—and is substantially higher in those affected by colorectal and ovarian cancer (10%–28% and 20%–50% per year, respectively). Breast cancer and melanoma are the most common non–intra-abdominal cancers to cause bowel obstruction, and this typically presents as diffuse peritoneal carcinomatosis.

Obstruction can occur anywhere along the gastrointestinal tract. In some cases, this represents the initial symptom of the underlying malignancy, whereas in others it develops during the course of disease. A recent consensus conference defined a malignant bowel obstruction (MBO) as one of the following: clinical evidence of bowel obstruction (by history, physical, and/or radiologic examination); bowel obstruction beyond the ligament of Treitz, in the setting of a diagnosis of intra-abdominal cancer with incurable disease; or a diagnosis of non–intra-abdominal primary cancer with clear intraperitoneal disease. Many treatment options exist for these patients, and because MBOs rarely need to be managed emergently, the treatment team, patient, and family have time to discuss these decisions in the context of the patient’s goals of care and underlying burden of disease.

Obstruction is categorized as either partial or complete. In partial obstruction, gas or liquid stool can pass through the point of intestinal narrowing. With complete obstruction, no substance can pass. Such obstructions can be secondary to mechanical or functional pathology. Mechanical obstructions are characterized as intraluminal (enlarging primary or secondary tumor), intramural (infiltration of the bowel wall), or extramural (extrinsic compression from masses, nodes, or adhesions) (Fig. 1). Functional obstruction is characterized by an adynamic ileus. This can result from tumor infiltration of the mesentery that affects the bowel wall muscle and nerves, or it can be induced by the many pain medications used by this population. Ileus may also result from a paraneoplastic neuropathy, particularly in patients with lung cancer.

In the setting of a mechanical obstruction, distinction should be made between simple and closed-loop obstructions. Simple obstructions result when the intestinal lumen is partially or completely occluded at one or more points, with resultant proximal distention and distal decompression of the intestine. In contrast, closed-loop obstructions result from two sequential occlusions. In a healthy population, this is typically a result of an incarcerated hernia or volvulus. These obstructions can rapidly dilate and progress to strangulation and compromise the mesenteric vascular supply.
which results in ischemia. Strangulated closed-loop obstructions are surgical emerg-
ecies. If left untreated, these types of obstructions can have mortality rates of
20% to 30%.

The following sections address etiologies that can precipitate malignant small- and
large-bowel obstructions. In each section, the authors have highlighted the underlying
pathophysiology, differences in patient presentations, and management options.

**Small Bowel Obstruction**

In Western countries, the most common causes of small-bowel obstruction (SBO) are
postsurgical adhesions (74%), Crohn’s disease (7%), and neoplasm (5%). Determining
the etiology of postsurgical SBO in patients with cancer can be particularly
challenging. SBOs can be caused by intrinsic narrowing from the malignancy or
extrinsic compression of the bowel by tumor or adhesions. Once an obstruction is
present, intestinal secretions and swallowed air accumulate, resulting in dilation of
the proximal small bowel. This stretch stimulates additional secretory activity and
microvascular changes in the bowel wall, with resultant translocation of bacteria to
the mesenteric lymph nodes.

The most common presenting symptoms of SBO are abdominal cramps, nausea,
vomiting, and abdominal distention. A source of continuous pain may point to the
location of the tumor, but patients may also have diffuse, nonlocalized signs. The
time course of symptoms, character of the emesis, and degree of distention also
provide helpful historical clues. For example, a rapid course, bilious emesis, and
minimal distention point toward a high-grade obstruction, whereas a more insidious
course, nonbilious emesis, and marked abdominal distension suggest a more distal
SBO or large-bowel obstruction (LBO). Passage of flatus or loose stools may
confound a history of obstruction as these can result from peristalsis above and below
the obstruction early on in the course of disease.

Although the history, clinical examination, and screening laboratory tests may be
helpful in the management of SBO, imaging is the cornerstone of establishing the
definitive diagnosis. Imaging helps determine the level of obstruction, potential for
concurrent strangulation, and the need for immediate operative intervention.

Plain radiographs using two views (flat and upright) have a sensitivity of 66% for
detecting an SBO, with low specificities affected by the inability of this modality
to distinguish between SBO and etiologies causing functional or mechanical
Moreover, abdominal plain films, in conjunction with clinical and laboratory findings, cannot reliably distinguish between a simple and strangulated obstruction. Despite these limitations, these images can be rapidly obtained, performed at the bedside, and can guide the early management and subsequent imaging. Images that show dilated gas or fluid-filled loops of small bowel with a gasless colon are pathognomonic of SBO (Fig. 2). Additionally, the presence of air-fluid differential height in the same small-bowel loop and a mean level width greater than 2.5 cm suggest a high-grade or complete SBO.

Early studies of CT performed to assess for SBO found 90% to 96% sensitivity and 96% to 100% specificity. Subsequent work questioned these results, with substantially lower sensitivity (63%) and specificity (78%) when the study population had a higher percentage of low-grade SBO. Further complicating this data, most of these earlier studies were performed with mono-slice CT, in contrast to the multidetector machines currently in use. Despite these conflicting results, CT offers distinct advantages over conventional radiography, especially in patients with underlying malignancy. First, CT can determine the presence of a closed-loop obstruction and strangulation. A negative predictive value of 89% makes this a useful test for patients in whom a conservative management strategy is planned. Second, multiplanar CT can more accurately visualize adjacent anatomy, define the point of obstruction (the so-called “transition point”), and identify intestinal pneumatosis. Lastly, recent evidence suggests that a CT augmented with enteroclysis—a process in which water-soluble contrast is infused by way of a catheter into the duodenum or proximal small bowel—offers a greater sensitivity and specificity for partial obstructions (89% and 100%,

![Small bowel obstruction radiograph.](From MedicalFinals.co.uk. Available at: www.medicalfinals.co.uk. Accessed December 12, 2008; with permission.)
respectively) and allows for greater differentiation between malignant and benign causes of SBO.23

In past decades, amid concern for strangulation and the limited diagnostic yield of abdominal radiographs, SBO was routinely managed operatively, following the adage of “never let the sun set or rise on an SBO”.40 Management has evolved in the setting of improved imaging modalities, and it is increasingly recognized that a conservative approach employing fluid resuscitation and nasointestinal suction may obviate surgery.39,41 This is encouraging for patients with MBOs, because surgical interventions carry a high operative mortality (5%–32%, often due to progression of disease), involve high morbidity (42%), and are often unsuccessful, with reobstruction rates of 10% to 50%.19,42–48 Conversely, among patients who develop a malignant SBO, 26% are because of underlying adhesions. In one series, this subgroup of patients lived longer and had more effective relief when they were managed surgically.49

A conservative approach begins with nasointestinal decompression as a means to relieve the small bowel of fluid accumulation. Typically, this is achieved by way of a nasogastric tube. Some investigators believe that more effective decompression can be achieved if the tube is advanced beyond the pylorus, which may result in better outcomes.15,50 Placement of these long intestinal tubes is more challenging than the typically used Salem sump nasogastric tube (Sherwood Medical, St Louis, MO, USA) and requires fluoroscopy, which may limit their widespread application in the ED.51

In the context of a nonsurgical management plan, pharmacotherapy may be a useful adjunct to bowel rest. Somatostatin analogs (octreotide) and anticholinergic agents (scopolamine) have been employed to reduce gastrointestinal secretions and lengthen small bowel transit time, with marked symptomatic relief and resolution of symptoms.52–56 Because octreotide has been shown to dramatically reduce the rate of nausea and vomiting with few side effects, and because complete relief of these symptoms is rare with typical antiemetics, some authors advocate the early use of this agent.10 The dose of octreotide used to manage MBO in small case series has ranged from 0.1 to 1.2 mg/d, administered by either a continuous or bolus subcutaneous infusion.6 Corticosteroids have not been shown to definitely affect the relief of MBOs, although they can act to treat obstruction-related pain.44 Corticosteroids should thus be reserved for patients in the terminal stages of their disease and be administered after consultation with the oncologist.10

Because of high mortality rates associated with nonsurgical care, the diagnosis of a closed-loop obstruction or strangulation should prompt the consideration of immediate surgical intervention. Additionally, if conservative management of a simple SBO fails, surgery may become a necessary next step. Laparoscopic adhesion lysis has grown in popularity as a minimally invasive technique to relieve SBO, with success rates of 40% to 70% in the general population.16 In patients with malignant SBO, CT imaging may be helpful to define the etiology of obstruction (tumor vs adhesions) and to map the location and type of adhesions.16 This may ultimately dictate whether a surgical approach is at all possible, and if so, whether the lesions are amenable to laparoscopic reduction. Palliative procedures such as ostomies, stenting, debulking, and intraperitoneal chemotherapy may also be considered.10

**Large Bowel Obstruction**

LBOs are much less common than SBO. However, when they are encountered, their most common cause is an underlying malignancy.57 Other etiologies include diverticulitis and volvulus of the sigmoid colon or cecum. In the setting of a malignant LBO, the cause is typically an intraluminal tumor growth, but it can also be the result of intussusception (discussed in a later section). Up to 20% of patients with colorectal cancer
develop an LBO, which can have a substantial morbidity and mortality. Postoperatively, up to 25% of patients with a colorectal cancer resection develop an LBO.\textsuperscript{58–61}

Similar to SBO, mechanical obstruction of the large bowel causes bowel dilatation above the site of obstruction. This precipitates mucosal edema, impaired vascular flow to the bowel wall, increased mucosal permeability, and subsequent bacterial translocation. A nonpatent ileocecal valve can create a second anatomic barrier, thus setting the stage for a closed-loop obstruction. It is essential for the practitioner to distinguish between LBO and non-obstructive causes of large bowel dilation, such as a toxic megacolon. The latter refers to dilation of the colon associated with acute colitis, either segmental or diffuse, without associated obstruction. A wide range of etiologies can result in acute colitis: inflammation, ischemia, infection, or radiation.

Symptoms of LBO typically follow an indolent course, with progression over several days. Initially, lower abdominal or diffuse colicky pain is accompanied by abdominal distention and changes in bowel habits (stool caliber changes or obstipation). Depending on the patency of the ileocecal valve, these symptoms can eventually progress to nausea and vomiting.\textsuperscript{62} Rarely, this emesis is feculent, a result of bacterial overgrowth in the stagnant fluid proximal to the obstruction.\textsuperscript{62} Evidence of systemic symptoms, such as fever or tachycardia, and laboratory abnormalities, such as a white blood cell count over \(20,000/\text{mm}^3\), may suggest the presence of a gangrenous bowel or perforation.\textsuperscript{57} However, immunocompromised patients may continue to have unimpressive abdominal examinations, even in the presence of these abdominal catastrophes.

Abdominal radiographs, when performed for evaluation of LBO, have a sensitivity and specificity of 84% and 72%, respectively (Fig. 3).\textsuperscript{63} In patients who are mobile, the addition of a contrast enema plain radiograph improves the sensitivity (96%) and specificity (98%) of this study to detect a mechanical obstruction.\textsuperscript{63} These studies do not, however, evaluate the viability of the proximal bowel, determine the extent of the primary malignancy or distant metastases, or significantly affect subsequent procedural planning.\textsuperscript{58}

![Fig. 3. Large bowel obstruction radiograph. (From Ho V, Lee YC. Acute colonic pseudo-obstruction in an elderly alcoholic man. Internet J Gastroenterol 2007;5(2); with permission.)](image-url)
Early studies using CT to detect LBO found that it was able to confirm the diagnosis in over 90% of cases, with one study citing a sensitivity of 96% and specificity of 93%. This same study found an improved diagnostic accuracy, sensitivity, and negative predictive value of CT when compared to the contrast enema outlined earlier. The use of multidetector CT, with improved spatial resolution and multiplanar images, likely improves its diagnostic accuracy for LBO, although evidence to support this is scarce. CT also enables the clinician to accurately exclude closed-loop obstructions or strangulation, evaluate for the nonobstructive causes of colonic dilation outlined earlier, and assess for nonmalignant causes of LBO such as volvulus and diverticulitis.

Once identified, malignant LBOs necessitate prompt intervention. Historically, this has involved operative management, with approximately 85% of emergency large bowel surgery performed to relieve malignant LBO. These obstructions can be addressed by either a palliative stoma or a rectosigmoid resection with accompanying colostomy, although these operations portend frequent complications and high mortality rates (8.8%–27%). Recent advances in endoscopy have established a role for colonic stenting in the management of malignant LBO, either as a palliative measure or as a bridge toward definitive operative management in patients with resectable disease. Stenting carries the risk of perforation (3%–4%), migration (10%–11%), and reobstruction (7%–10%). A meta-analysis that compared operative versus endoscopic management of malignant LBO found that colonic stents were associated with significant reductions in length of hospital stay, mortality rate, eventual need for a palliative stoma, and medical complications. Moreover, mortality rates were similar between patients who underwent emergency surgery and those who were stented in anticipation of an elective bowel resection, although there was substantial heterogeneity among the two groups. A prospective trial that compares these two interventions is ongoing (http://www.crtrial.com).

Gastric Outlet Obstruction
Gastric outlet obstruction (GOO) results from an underlying malignancy in 63% of cases. Typically, cancer involving organs of the lesser sac results in intrinsic or extrinsic obstruction of the adjacent pyloric channel or duodenum. This obstruction may result from a combination of local infiltration, edema, and adhesions. The symptoms of GOO may be intermittent until occlusion is complete. Pancreatic cancer is the most common malignancy to cause GOO (10%–20%). Other etiologies that cause GOO include ampullary cancer, duodenal cancer, cholangiocarcinoma, gastric cancer, and metastases to the gastric outlet from lung and breast cancer.

Early symptoms of GOO may include gastroesophageal reflux, early satiety, and weight loss. Eventually, patients progress to nausea and vomiting, usually nonbilious with undigested food particles, along with abdominal pain and distention. Although nausea and vomiting are frequent complaints in patients with any underlying malignancy, especially in the context of ongoing chemotherapy, this sequence of symptoms should also prompt consideration of GOO.

Although it is difficult to definitively establish the diagnosis of GOO in the ED, patients can be managed symptomatically with fluid resuscitation and gastric decompression. Gastrojejunostomy remains the definitive surgical treatment of choice, although self-expandable metallic stents have become a viable nonsurgical alternative, with relief of obstruction in more than 90% of cases. Studies that compare these two interventions have shown equivalent survival rates, although patients who received endoscopic stents had shorter hospital stays, lower costs, and an improved ability to consume food orally. Patients managed surgically, however, had
a reduced need for reintervention, a result attributed to delayed stent occlusion.\textsuperscript{81,82} As an additional option, gastric venting can be achieved by way of a percutaneously placed gastrostomy tube.\textsuperscript{6}

**Malignant Intussusception**

Intussusception occurs in all age groups, but is primarily a disease of infancy and early childhood. Overall, only 5\% of intussusceptions occur in adults, and intussusceptions account for approximately 1\% to 5\% of adult SBOs.\textsuperscript{83,84} Childhood causes tend to be idiopathic, whereas a mechanical cause is present in more than 90\% of adult cases. Solid tumors, benign and malignant, act as the lead point in over 65\% of adult cases.\textsuperscript{83,85} Intussusception results when a segment of bowel lumen that either contains a mass lesion or has altered peristaltic activity telescopes into an adjacent segment. This results in obstruction and ischemic injury to the intussuscepting segment.\textsuperscript{84} A neoplastic mass must be strongly considered in children older than 4 years and adults who present with intussusception.

Patients with intussusception have acute, subacute, or chronic nonspecific symptoms. In small studies, the most common complaint reported is intermittent abdominal cramping (79\%), followed by obstructive signs, such as nausea, vomiting, and abdominal distention.\textsuperscript{83,84} Plain radiographic evaluation of the abdomen is similarly nonspecific. The abdominal radiograph may suggest partial or complete bowel obstruction, although occasionally a soft-tissue density outlined by two strips of air may be seen.\textsuperscript{83,84,86} The use of CT in adults has been recently advocated to better differentiate an associated mass and rule out other etiologies. Characteristic imaging findings include the “target sign” or “sausage-shaped appearance.”\textsuperscript{83,85} Management of adult intussusceptions is dictated by their location. Pure colonic intussusceptions are typically resected because of their high malignant potential, whereas ileocolic lesions may be approached with intraoperative colonoscopy to identify the lesion before intervention and potentially avoid resection.\textsuperscript{84,86}

**PERFORATION**

MBOs, transmural erosion of primary gastrointestinal cancers, metastatic lesions to the gut, and atypical infections have the potential to cause bowel perforation and peritonitis. The clinical severity of these perforations and therapeutic options for treatment are affected by the location, involvement of adjacent structures, and the time to patient presentation. Intra-abdominal perforations are categorized as either free, when bowel contents are spilled into the abdominal cavity, or contained, if contiguous organs wall off the area. In an immunocompromised population, over 90\% of colonic perforations result in pneumoperitoneum because of these patients’ inability to localize infection and the late onset of presentation.\textsuperscript{2}

The differential for peritonitis in this population is shaped by the patient’s underlying malignancy and degree of immunosuppression. Common causes of colonic perforations in immunocompromised patients include diverticular disease, ischemic colitis, fecal impaction, neutropenic enterocolitis, and opportunistic infections such as cytomegalovirus (CMV) and Candida.\textsuperscript{2,87,88} Perforations secondary to CMV have also been found in the ileum and jejunum among patients undergoing chemotherapy.\textsuperscript{89–92}

CT has the ability to demonstrate small amounts of free air, pneumatosis intestinalis, portal venous air, and the site of intestinal obstruction.\textsuperscript{2,93} Once perforation is established, broad-spectrum antibiotics along with rapid surgical consultation should be initiated with likely operative management.
PATHOLOGY UNIQUE TO PROFOUND IMMUNOSUPPRESSION

All patients with an underlying malignancy who present to the ED should be considered immunosuppressed. The degree to which this immunosuppression affects their clinical presentation is dependent upon whether they recently received chemotherapy, their current absolute neutrophil count, and associated medical problems. Patients that are categorized as severely immunosuppressed (see group II in Table 1) require a special consideration of unique diagnoses. The most common cause of an acute abdomen in neutropenic patients, neutropenic enterocolitis, is discussed in detail elsewhere in this issue. Additional pathologies are outlined in Table 2.

Neutropenic Enterocolitis

Neutropenic enterocolitis, or typhlitis, is a necrotizing inflammation of the cecum that is found almost exclusively in patients who are immunocompromised. This entity has received a multitude of names: neutropenic colitis, necrotizing enterocolitis, ileocecal syndrome, and cecitis.94–96 The incidence of this disease among adults varies widely in the literature (0.8%–26%), likely due to the heterogeneity of the diagnostic criteria.97 When identified, this diagnosis carries a mortality rate of 50% and higher.97–103 Among patients with solid or hematologic malignancies, typhlitis typically manifests in the setting of chemotherapy-induced neutropenia or bone-marrow transplantation, and it has emerged as the most common cause of the acute abdomen in the neutropenic cancer patient.2 Case reports also cite neutropenic enterocolitis as a presenting symptom in patients with acute myelogenous and acute lymphoblastic leukemia, before the onset of chemotherapy.104–109 Moreover, it has also been described in patients with AIDS, as well as patients who require immunosuppression after organ transplantation.110,111 Despite its name, patients can develop neutropenic enterocolitis in the absence of neutropenia.112–114

The etiology and pathogenesis of this syndrome remain poorly understood. It has been postulated that the disease is caused by the inability to maintain an intact intestinal mucosa. This lack of mucosal integrity likely results from a combination of neutropenia, impaired host defenses, and direct injury from chemotherapeutic agents.111

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Additional presentations unique to underlying malignancy or immunocompromise</th>
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<tbody>
<tr>
<td>Disease State</td>
<td>Manifestations</td>
</tr>
<tr>
<td>CMV</td>
<td>GI tract mucosal ulcers (mouth to rectum), commonly with secondary bleeding that can progress to perforation.</td>
</tr>
<tr>
<td>Varicella (diffuse &amp; primary)</td>
<td>Acute abdomen can occur without cutaneous findings. Typically in stem-cell transplant patient, associated with hyponatremia secondary to SIADH. Treated with acyclovir and IVIG.</td>
</tr>
<tr>
<td>GVHD</td>
<td>Skin (rash/dermatitis), liver (hepatitis/jaundice), GI tract (abdominal pain/diarrhea). Findings can occur together or in isolation.</td>
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</table>

Abbreviations: GI, gastrointestinal; GVHD, graft-versus-host disease; IVIG, intravenous immune globulin; SIADH, syndrome of inappropriate antidiuretic hormone.
Transmucosal migration of bacteria causes systemic bacteremia, endotoxin production, mucosal or transmural necrosis, and focal hemorrhage.\textsuperscript{94,110}

There is considerable variation in presentation among patients with neutropenic enterocolitis. The most common symptoms are fever, right lower quadrant abdominal pain, nausea, and vomiting. Computed tomography is the imaging modality of choice because this constellation of complaints poses a fairly wide differential. Ultrasound and abdominal plain films may provide clues to this diagnosis, but have higher false negative rates.\textsuperscript{115} Typical CT findings (Fig. 4) include circumferential cecal wall thickening; dilated, fluid-filled cecum; and adjacent mesenteric fat stranding.\textsuperscript{94} The degree of bowel-wall thickening may correlate with prognosis, with one retrospective study showing a mortality of 60\% if the bowel wall was 10 mm or more by ultrasound, and only 4.2\% if 10 mm or less.\textsuperscript{116} Focal findings of high attenuation in the cecal wall may represent hemorrhage.

Neutropenic enterocolitis is an uncommon diagnosis, and there are no randomized prospective trials to guide management. Most authors advocate broad-spectrum antibiotics, bowel rest, aggressive nutritional supplementation, and correction of underlying cytopenias and coagulopathies.\textsuperscript{94,104} If the patient is neutropenic, administration of granulocyte colony-stimulating factor may provide benefit. A more detailed review of neutropenic enterocolitis is presented elsewhere in this issue.

**ETIOLOGIES UNIQUE TO PATIENTS WITH CANCER**

**Malignant Ascites**

Malignant ascites is defined as the collection of fluid containing cancerous cells in the abdomen. Malignant ascites can occur from direct invasion of the peritoneum (peritoneal carcinomatosis), or as a result of venous or lymphatic obstruction.\textsuperscript{117} Ovarian cancer is the most common malignancy to cause ascites, occurring in 37\% of all reported cases. Because of this, malignant ascites is twice as likely in females as males (67\% vs 33\%).\textsuperscript{118,119} Additional malignancies complicated by ascites include, but are not limited to, pancreaticobiliary, gastric, esophageal, and colorectal cancers. The pathophysiology of ascitic fluid accumulation is multifactorial and may result from venous and lymphatic obstruction, oncotic shifts, or enhanced vascular permeability as a result of diffuse cytokine release.\textsuperscript{120}

Patients with malignant ascites can present with abdominal distention, pain, dyspnea, nausea, anorexia, and vomiting. In more than 50\% of patients ultimately

![Fig. 4. Computed tomography image of neutropenic enterocolitis. (From Lodhawala T, Shaikh A, Carmosino L. Gastroenterology photo quiz. Resid Staff Physician 2007;53(1), with permission.)](image-url)
diagnosed with malignant ascites, this fluid accumulation caused the first presenting signs and symptoms of their underlying malignancy. Because of the many potential etiologies that can cause ascites, paracentesis is necessary to establish a malignant source. In these cases, cytology is diagnostic in 50% to 60% of cases, with sensitivities of 58% to 75%.

Management strategies for malignant ascites are directed toward symptomatic relief, similar to the treatment of patients with nonmalignant ascites. Initial interventions include diuretics and therapeutic paracentesis. When these measures are unsuccessful, a peritoneal drain can be placed, with relief in 83% to 100% of cases. A peritoneovenous shunt (PVS) incorporates 1-way valves allowing flow from the peritoneum to the vena cava and is a more invasive alternative to the extraperitoneal drain. Although there is little data to compare these two interventions, PVS has been shown to have a higher rate of patency at 72 days. There is a theoretical risk of disseminated intravascular coagulation in patients with PVS, because of shunting of cancerous cells into the vena cava.

Patients with malignant ascites are at risk for spontaneous bacterial peritonitis, although this risk is believed to be lower than in patients with nephrotic or cirrhotic ascites. That said, because of the concurrent immunosuppression, physicians should have a low threshold to perform diagnostic paracenteses as part of their infectious work-up. To address the malignant ascites itself, palliative intraperitoneal chemotherapy can be considered.

Radiation Enteritis

Radiation enteritis occurs in patients receiving irradiation of the abdomen or pelvis during the course of their cancer treatment. The disease is characterized by diffuse collagen deposition and progressive occlusive vasculitis that results in local narrowing of the intestinal lumen. These microvascular changes result in bowel dilation proximal to affected segments and mucosal damage, with secondary effects of bowel ulceration, perforation, and massive gastrointestinal bleeding. Pelvic irradiation, where the greatest volume of bowel is affected, is predominantly used in the treatment of gynecologic and urologic cancers. A recent United Kingdom study cited a 50% incidence of radiation-induced bowel damage among their cohort of 12,000 patients with pelvic cancer. Although only 4% to 8% of these patients had life-threatening complications, as many as 80% were chronically affected by malnutrition, fistula formation, and strictures.

The small bowel is particularly susceptible to radiation enteritis, especially at doses greater than 45 Gy. The most important factors to prevent both acute and chronic radiation-induced injury are the reduction of dose and field size. Once radiation enteritis is diagnosed, medical therapy can include 5-aminosalicylate, octreotide, and the administration of probiotics. These measures have been shown to be most effective in the acute phase. Unfortunately, few pharmacologic choices for chronic radiation enteritis have shown an established benefit. Ongoing research is evaluating cytokine levels as a means to predict which patients are most susceptible to radiation enteritis, manipulation of growth factors in formation of fibrotic tissue, and dietary supplementation.

Budd-Chiari Syndrome

The Budd-Chiari syndrome (BCS) describes the clinical manifestations of hepatic venous outflow obstruction at the level of the hepatic venules, large hepatic veins, inferior vena cava, or right atrium. These obstructive thromboses occur most frequently in the setting of an underlying hypercoagulable state and in rare cases...
can result from tumor invasion. The malignancies that most frequently precipitate BCS are hepatocellular, renal-cell, and adrenal carcinoma.\textsuperscript{130} The resultant venous stasis and hepatic congestion cause hypoxic parenchymal cell injury, centrilobular necrosis, and eventual cirrhosis.\textsuperscript{130} Unless hepatic congestion is relieved, obstruction progresses to liver failure and death.\textsuperscript{132}

Typical symptoms of the Budd-Chiari syndrome include ascites, hepatomegaly, and abdominal pain, with some cases progressing to jaundice, portal hypertension, and variceal bleeding.\textsuperscript{131} Coagulopathy, encephalopathy, and development of the hepatoportal syndrome all indicate a poor prognosis.\textsuperscript{130} The diagnosis can be established using ultrasound, CT, or MRI. If advanced imaging is inconclusive, a liver biopsy may be necessary.\textsuperscript{133,134} The sensitivity of Doppler ultrasonography for diagnosing BCS is 85% to 90%, and this should be the initial imaging technique performed when this diagnosis is entertained.\textsuperscript{135,136} Ultrasonography findings in BCS include “nonvisualization of hepatic veins, absence of flow in the hepatic vein, stenosis of or thrombosis within the hepatic veins, intrahepatic collaterals, and obstruction of the infrahepatic inferior vena cava”.\textsuperscript{135} CT and MRI offer the advantages of better identification of necrotic areas of the liver and improved detail of liver perfusion.\textsuperscript{135}

For relatively asymptomatic patients with BCS who have normal liver function tests, medical therapy with sodium restriction, diuretics, anticoagulation, and intermittent paracenteses can constitute the initial management strategy. In the setting of hepatocellular necrosis, however, patients need either surgical or percutaneous portosystemic shunting (mesoatrial, mesocaval, transjugular intrahepatic portosystemic shunting or hepatic vein angioplasty).\textsuperscript{132,137} Both treatments are typically followed by lifelong systemic anticoagulation.\textsuperscript{132} Fulminant or refractory cases of Budd-Chiari syndrome may require liver transplantation.

SUMMARY

Gastrointestinal complaints among patients with cancer can pose a diagnostic dilemma with significant rates of morbidity and mortality. Mass lesions or adhesions related to intra-abdominal malignancies place these patients at higher risk for bowel obstruction or perforation, while ongoing chemotherapy or functional immunosuppression can mask many of the findings among healthy patients. This affects the timing of patients’ presentation and their clinical examination in the ED. Moreover, in the setting of profound immunosuppression, a wide range of atypical infections and pathologies must be considered. Clinical vigilance by the practitioner is thus essential, with a low threshold to use advanced imaging modalities and careful consideration of this expanded differential.

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