CI

e-med
www.e-med.org.uk

REVIEW

MDCT of abdominopelvic oncologic emergencies

Sree Harsha Tirumani[a], Vijayanadh Ojili[b], Gowthaman Gunabushanam[c], Kedar N Chintapalli[b], John G Ryan[d], Caroline Reinhold[e]

[a]Department of Imaging, Dana Farber Cancer Institute/Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02215, USA; [b]Department of Radiology, University of Texas HSC at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229, USA; [c]Department of Diagnostic Radiology Yale University School of Medicine, 333 Cedar St, PO Box 208042, New Haven, CT 06520-8042, USA; [d]Department of Diagnostic Imaging, The Ottawa Hospital, Civic Campus, 1053 Carling Ave, Ottawa, ON K1Y 4E9, Canada; [e]Department of Diagnostic Radiology, Montreal General Hospital, McGill University Health Centre, 1645 Avenue Cedar, Montreal, QC H3G 1A4, Canada

Corresponding address: Vijayanadh Ojili, MD, Department of Radiology, University of Texas HSC at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229, USA.
Email: ojili@uthscsa.edu

Date accepted for publication 15 April 2013

Abstract

Acute complications arising in abdominopelvic malignancies represent a unique subset of patients presenting to the emergency room. The acute presentation can be due to complications occurring in the tumor itself or visceral or vascular structures harboring the tumor. Multidetector computed tomography (MDCT) is the investigation of choice in the workup of these patients and enables appropriate and timely management. Management of the complication depends primarily on the extent of the underlying malignancy and the involvement of other viscera. The purpose of this article is to depict the imaging features of these complications on MDCT.

Keywords: *Oncology; abdominopelvic malignancy; acute abdomen; MDCT.*

Introduction

Oncologic emergencies are potentially life-threatening conditions encountered in cancer patients related directly or indirectly to the underlying malignancy. These emergencies can result in rapid deterioration of the clinical course and are associated with significant morbidity and mortality. The main emphasis in the literature has always been on conditions like metabolic, cardiovascular, neurologic, infectious, hematologic and respiratory emergencies[1,2]. Acute abdomen in cancer patients, although less often discussed as a distinct entity, is unique and has significant diagnostic, therapeutic and prognostic implications. Malignant tumors, due to their ability to grow, invade surrounding viscera and metastasize to distant sites, can result in obstruction of hollow viscera, vessels or ducts, hemorrhage, thrombosis, rupture or infiltration of solid organs, all of which can present as abdominal pain. In addition, several other acute intra-abdominal pathologic conditions, which are routinely encountered in the general population, can be seen in this subset of patients.

Acute complications of abdominopelvic malignancies have often been described as individual entities. The lack of disease and organ specificity of these complications renders a comprehensive classification difficult and untenable. A broad classification that can guide management to some extent includes complications occurring within the tumor and complications occurring in other abdominopelvic viscera and vasculature due to tumor growth and invasion (Table 1). Acute complications occurring in the tumor may bring attention to the tumor for the first time, in some cases at an early stage in the disease process when the tumor is surgically resectable. Emergencies resulting from invasion of other viscera usually portend a poor prognosis due to advanced stage and include gastrointestinal, hepatobiliary, pancreatic, urinary and vascular complications. Radiologic investigations play a critical role in detecting these complications and enable prompt triage of the patients. Multidetector computed tomography (MDCT), the most widely available, easily accessible and comprehensive imaging technique in the emergency setting, is the

---

Table 1 **Summary of oncologic emergencies in the abdomen and pelvis**

Oncologic emergencies	Causes	MDCT features	Management
Intratumoral hemorrhage/rupture	Vascular tumors (HCC/RCC) Bulky neoplasms Anticoagulation, biopsy or chemoembolization	Intratumoral or subcapsular bleed Frank hemoperitoneum High-attenuation (30–40 HU) intra- or peritumoral and/or intraperitoneal fluid	Conservative Embolization Surgical management
Torsion	Ovarian tumors GIST	Twisted vascular pedicle with whirlpool sign Decreased tumor enhancement Intratumoral hemorrhage/rupture Peritumoral stranding	Surgical management
Infection/inflammation	Endometrial cancer Cervical cancer Rectal cancer Appendicular mucinous neoplasms	Abscesses Fistulas Pyometra Perforartion Pseudomyxoma peritonei	Antibiotics Drainage of abscess Defunctioning stoma Surgical resection
Gastrointestinal complications (bowel obstruction, ischemia, perforation)	Gastric cancer Pancreatoduodenal cancers Small bowel metastases Colon cancer Peritoneal carcinomatosis	Gastric outlet obstruction Small/large bowel obstruction Enhancing serosal/mural/intraluminal deposits Intussusception Bowel ischemia Perforation Pneumatosis intestinalis	Endoluminal stents Surgical management
Hepatic complications (acute liver failure)	Budd-Chiari syndrome (HCC, RCC, ACC) Tumoral infiltration Malignant biliary obstruction	Tumor thrombus in IVC/hepatic veins Extensive hepatic metastases Biliary dilatation	Chemotherapy Recanalization of the IVC
Biliary complications (acute cholangitis)	Malignant biliary obstruction	Thickened biliary walls Cholangitic abscesses	Antibiotics Biliary drainage
Pancreatic complications (acute pancreatitis)	Pancreatic cancer Metastases (lung, breast, RCC) Malignant hypercalcemia	Enlarged pancreas Peripancreatic stranding Metastatic deposits	Conservative management
Urinary complications (acute renal failure)	Tumor infiltration (lymphoma, leukemia, metastases from solid tumors)	Renal enlargement Solid masses	Chemotherapy
	Postrenal causes (cancers of cervix, prostate, urinary bladder, colon)	Bladder outlet obstruction Hydroureteronephrosis	Urinary diversion
Vascular complications (thrombosis)	Venous thrombosis (HCC, RCC, lymphoma, pancreatic cancer, metastatic adenopathy) Arterial thrombosis (pancreatic cancer)	Budd-Chiari syndrome Lower extremity deep venous thrombosis Pulmonary embolism End-organ ischemia	Anticoagulation Vascular recanalization

key imaging modality used in the workup of these patients. In this article, we present a comprehensive review of the non-metabolic, non–treatment-related oncologic emergencies of the abdomen and pelvis with emphasis on imaging features on MDCT.

Acute intratumoral hemorrhage and rupture

Malignant tumors such as hepatocellular carcinoma (HCC) and renal cell carcinoma (RCC) are known for high vascularity, which predisposes them to spontaneous intra-tumoral hemorrhage and rupture (Fig. 1). HCC can be complicated by life-threatening rupture and intraperitoneal hemorrhage in 3–15% cases resulting in about 70% mortality[3]. Rupture is more likely when HCC occurs in the peripheral or subcapsular location in a cirrhotic liver. Spontaneous renal and perirenal hemorrhage, also referred to as Wunderlich syndrome, complicates 0.3–1.4% of cases of RCC, most commonly the clear cell variant[4]. The mechanism of rupture in these tumors is not clearly known, but postulated theories
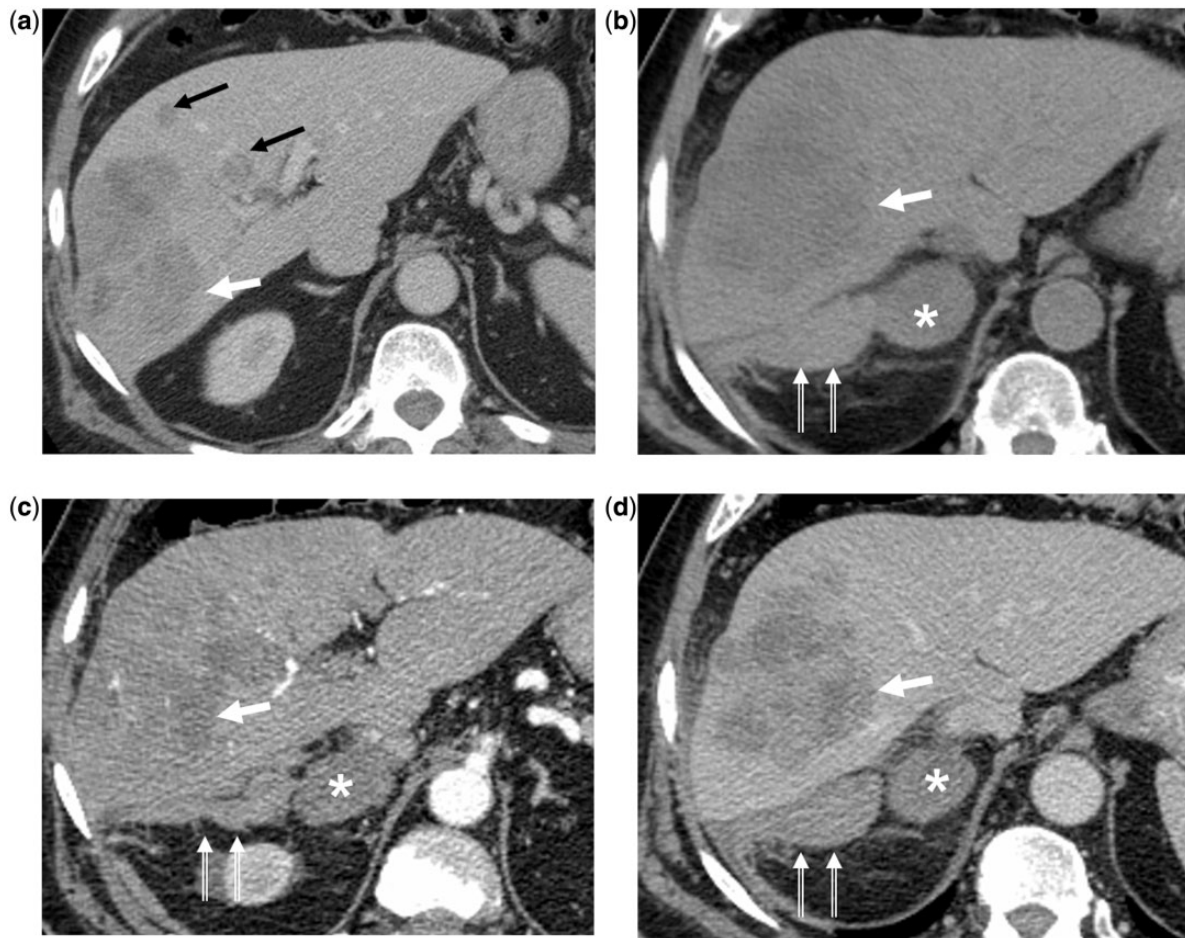
Figure 1 An 82-year-old man with ruptured HCC. (a) Axial contrast-enhanced CT image of the liver demonstrates a large heterogeneous liver mass (thick arrow) with satellite lesions (black arrows). (b,c,d) Six months later the patient presented to the emergency department with acute abdominal pain. Axial unenhanced (b), arterial (c) and venous (d) phase contrast-enhanced CT images of the liver demonstrate a high-attenuation perihepatic hematoma (double-line arrows) with stranding, which is suggestive of acute rupture of the hepatocellular carcinoma (thick arrows). Note the new right adrenal metastasis (*). The patient was managed conservatively.

include thrombosis of draining veins, which results in acute increase in the intratumoral pressure, fragile neovascular channels, rapid tumor growth and trivial trauma[5]. Gastrointestinal stromal tumors (GISTs) can be complicated by rupture and intraperitoneal hemorrhage either spontaneously or following trivial trauma[6] (Fig. 2). Bulky neoplasms like ovarian tumors[6], although not highly vascular, may rupture due to their size[7] (Fig. 3). Rupture of ovarian dermoid occurs in 1–4% cases resulting in acute peritonitis[8]. Hepatic metastases from melanoma, lung cancer, renal carcinoma and choriocarcinoma can be complicated by hemorrhage and rupture[9,10]. Iatrogenic causes such as systemic anticoagulation, biopsy or chemoembolization can occasionally result in intratumoral hemorrhage and rupture.

Rupture of a malignant neoplasm is a catastrophic emergency due to the risk of rapid exsanguination. Clinically, patients present with acute abdominal pain

and shock. The Lenk triad in Wunderlich syndrome refers to acute flank pain, shock and palpable flank mass[11]. Although ultrasonography detects hemoperitoneum, MDCT allows more comprehensive assessment by detecting the site and cause of hemorrhage, quantifying the amount of hemorrhage and detecting active bleeding, thereby triaging patients who need emergent care. The spectrum of findings on MDCT can range from minimal intratumoral or subcapsular bleed to frank hemoperitoneum[12] (Figs. 1–3). Non-enhanced computed tomography (CT) demonstrates acute hemorrhage as high-attenuation (30–40 HU) intratumoral or peritumoral and intraperitoneal fluid, usually in dependent areas such as the Morrison pouch and the pelvis (Fig. 1). In cases of non-traumatic hemoperitoneum, the site of hemorrhage can be identified by the presence of high-attenuation hematoma (45–70 HU) in the vicinity of an organ, referred to as the sentinel clot sign[13] (Figs. 2, 3).
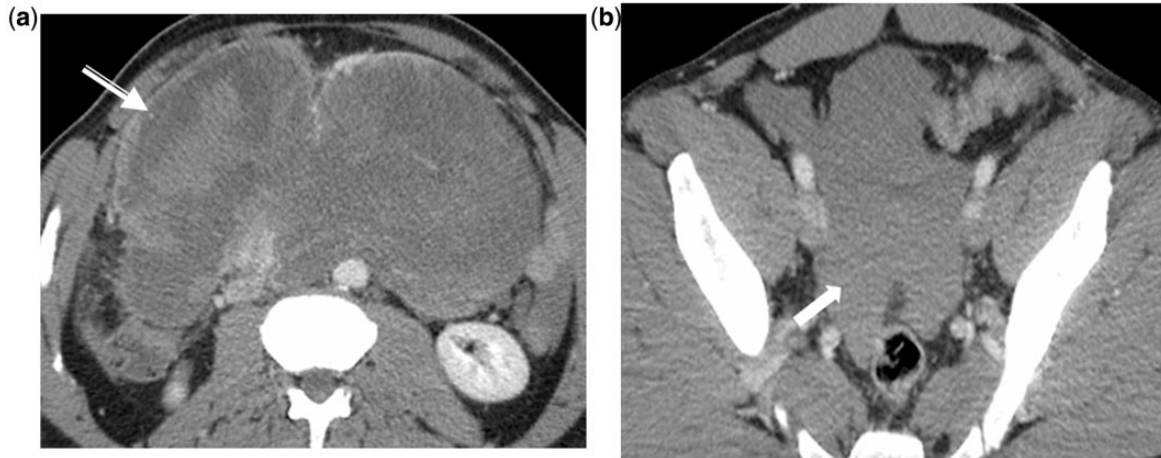
Figure 2 **A 65-year-old man with ruptured GIST. (a,b) Axial contrast-enhanced CT images of the abdomen (a) and pelvis (b) demonstrate a large heterogeneous upper abdominal mass (asterisk) with discontinuity of the tumor outline (double-lined arrow, a) along the right lateral aspect suggestive of tumor rupture. Note the hemoperitoneum in both flanks and pelvis (thick arrow, b). The patient was managed nonoperatively due to the presence of liver metastases (not shown). Subsequent biopsy revealed a GIST. The organ of origin of the tumor was not conclusive.**
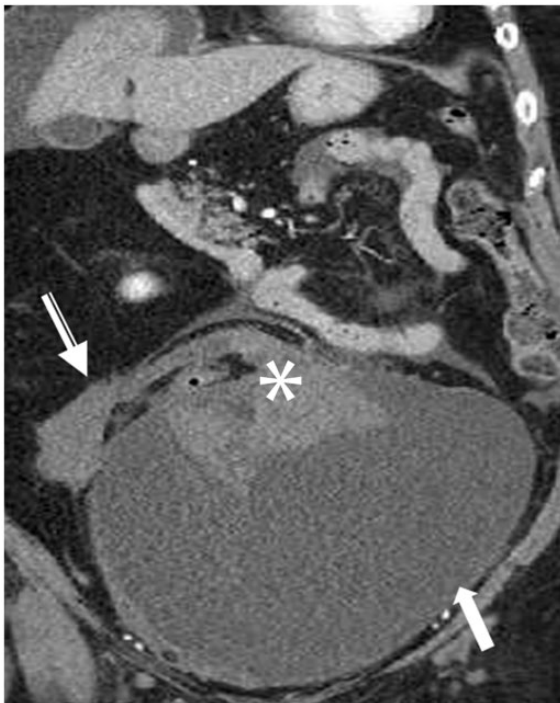

Figure 3 **A 43-year-old woman with rupture of a cystic ovarian neoplasm. Coronal reformatted contrast-enhanced CT image demonstrates a complex cystic abdominopelvic mass (arrow) with thin septations and a high-attenuation region in the superior aspect of the tumor (asterisk) suggestive of hemorrhage into a cystic ovarian neoplasm. In addition, there was high-attenuation fluid in the peritoneal cavity (double-lined arrow) suggestive of hemoperitoneum due to intraperitoneal rupture of the cystic ovarian neoplasm. Histopathology revealed ruptured mucinous cystadenocarcinoma of the ovary.**

The ruptured tumor is usually hypodense on the unenhanced CT image and is seen as an enhancing mass within the hematoma after contrast administration. Occasionally, however, it may be difficult to delineate the tumor in the acute phase. The detection rates of underlying masses in Wunderlich syndrome in the acute phase can be as low as 57%[4]. In these cases with no underlying cause for the spontaneous hemorrhage, repeat imaging in 2–3 months can demonstrate a mass. Rupture without hemorrhage is seen on imaging as discontinuity of the tumor capsule. The initial management of acute tumor rupture with intraperitoneal bleed revolves around resuscitation and restoration of circulatory volume. Subsequent management depends on the presence of ongoing hemorrhage, the underlying neoplasm and the presence of comorbidities. Hemodynamically stable patients and patients with inoperable tumors can be observed with careful monitoring of vital signs. Hemostasis can be achieved in patients with ongoing bleeding by angioembolization, surgical ligation of bleeding vessels or surgical resection of the tumor. In the case of ruptured HCC, the success rate of transarterial embolization in the acute phase is about 53–100%[14]. For definitive management, staged liver resection has a better outcome than emergency liver resection. Definitive treatment of ruptured RCC after achieving hemostasis depends on the stage of the tumor[4].

Acute torsion

Acute torsion is likely to occur in mobile organs and pedunculated neoplasms and results in acute devascularization of the tumor with the risk of hemorrhage and rupture. Torsion is most often encountered in ovarian
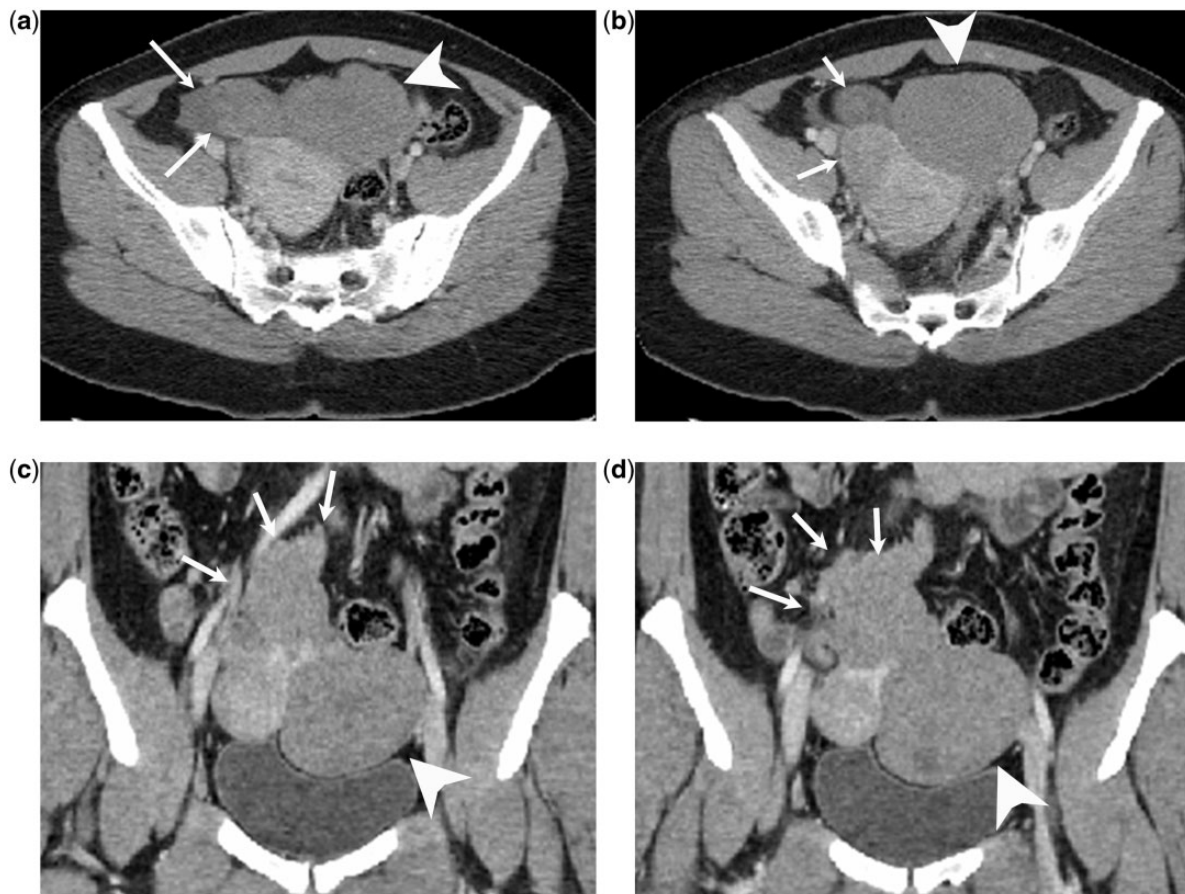
Figure 4 **A 51-year-old woman with torsion of a malignant ovarian tumor, presenting with a right lower quadrant mass. (a,b) Axial and (c,d) coronal reformatted contrast-enhanced CT images of the pelvis demonstrate a heterogeneous, solid mass (arrowhead) abutting the left anterosuperior aspect of the uterus and extending into the right lower quadrant with a tubular configuration (arrows) contiguous with the right lateral aspect of the uterus. There is decreased enhancement of the entire mass. The left ovary was seen separately (not shown). Emergent laparotomy confirmed a right tuboovarian mass with torsion, hemorrhagic infarction and necrosis. Histopathology revealed a necrotic ovarian tumor with malignant cells.**

tumors. Although teratomas are the most common ovarian tumors to undergo torsion, malignant ovarian tumors are also at risk for torsion[8,15] (Fig. 4). In a study of 135 cases of adnexal torsion, Lee et al.[16] found a malignant ovarian tumor in 15% of cases. In the pediatric age group and in postmenopausal women, torsion can complicate malignant ovarian tumors[17,18]. In a study of 27 postmenopausal patients with torsion, 22% were found to have underlying malignancy in the torted ovary[17]. The extreme mobility of the ovaries due to the long pedicle and the common ligamentous attachment of the fallopian tube predispose them to torsion (Fig. 4). Less commonly, tumors such as GISTs can undergo torsion due to their size[19]. The pathophysiologic consequences of torsion are venous and lymphatic obstruction, which cause massive intratumoral edema followed by progressive arterial obstruction resulting in hemorrhagic infarction and rupture of the tumor[8]. Clinically, torsion of

abdominopelvic tumors presents with sudden onset of abdominal pain mimicking a gamut of other non-neoplastic causes. Acute torsion of ovarian tumors can be confused clinically with acute appendicitis, diverticulitis, tuboovarian abscess, ectopic pregnancy and ruptured ovarian cyst. This can result in delay in diagnosis, especially in postmenopausal women who often have necrotic adnexa at the time of surgery[17].

Imaging plays a key role in the diagnosis of torsion. Ultrasonography is the initial investigation modality and can demonstrate the twisted vascular pedicle with decreased vascularity of the torted tumor on Doppler interrogation. However, findings at ultrasonography may be ambiguous and inconclusive depending on the degree of torsion and the experience of the operator[8]. In such cases, MDCT can confirm the torsion, detect complications and exclude other pathologic conditions. The presence of a twisted vascular pedicle with whirlpool
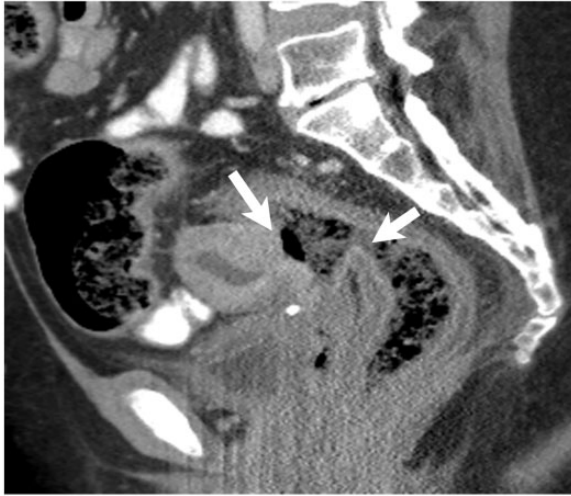
Figure 5 **A 61-year-old woman with cervical cancer complicated by a rectovaginal fistula. Sagittal reformatted contrast-enhanced CT image of the pelvis demonstrates heterogeneous cervical mass fistulizing with the rectum (arrows).**

Figure 6 **A 29-year-old woman with rectal cancer complicated by pelvic abscess. Axial contrast-enhanced CT image of the pelvis reveals asymmetric polypoidal thickening of the distal rectum (arrows) and a multiloculated abscess (asterisk) in the left ischiorectal fossa. Note the enlarged bilateral inguinal lymph nodes (double-line arrows). Diagnosis of perforated rectal cancer was confirmed subsequently at colonoscopy. Inguinal lymph node biopsy revealed no metastasis.**

sign, decreased enhancement of the tumor, intratumoral hemorrhage, rupture, peritumoral stranding, free fluid with or without hemoperitoneum are findings suggestive of torsion on MDCT (Fig. 4). Acute torsion of any tumor is a surgical emergency and requires resection of the tumor. In the case of ovarian tumors, adnexa-preserving surgery is preferred for benign tumors, whereas malignant tumors need a more aggressive surgical approach and staging laparotomy[15].

Acute infection and inflammation

Secondary infection and inflammation of a neoplasm is predisposed by necrosis in the tumor. Rapid tumor growth can result in spontaneous tumor necrosis, which may get secondarily infected by enteric pathogens either directly or hematogenously. Bulky uterine and cervical cancers frequently have superimposed infection due to the proximity to vaginal flora. Enterovaginal fistulas can complicate advanced cervical cancer or occur as a consequence of radiotherapy or surgery[20] (Fig. 5). Complex fistulas, especially rectovaginal fistulas, present with abscess formation. Pyometra refers to accumulation of pus within the uterine cavity resulting from cervical stenosis due to benign or malignant uterine neoplasms, surgery, radiotherapy or chronic cervicitis[21]. Uterine perforation is a devastating complication of pyometra. In a meta-analysis of 36 cases of perforated uterine pyometra, 31% cases were related to malignant causes[21]. Contained perforation of colorectal neoplasms may result in local abscess formation[22] (Fig. 6). Mucinous neoplasms of the appendix may get infected or inflamed with the risk of perforation and

pseudomyxomaperitonei[22]. Rarely, appendicitis may be the presenting manifestation of mucocele (Fig. 7).

Uncommonly, malignant neoplasms can mimic infectious conditions. In a search of a 12-year nationwide health registry in Taiwan, it was found that in up to 2% of cases, pyogenic abscess was the initial presentation of HCC[23]. Several cases of necrotic metastatic deposits from cervical cancers mimicking abscesses have been reported in the literature. Psoas abscess-like deposits from cervical cancer are known to occur in human immunodeficiency virus (HIV)-positive and even in HIV-negative women due to an aggressive growth pattern[24,25]. Clinically, infected neoplasms present with constitutional symptoms and systemic sepsis that can mask the underlying malignancy. Lower abdominal pain, purulent vaginal discharge and postmenopausal bleeding are the classic features seen in patients with pyometra[21]. MDCT with oral and intravenous contrast helps in prompt detection of the infected neoplasm and associated complications such as abscesses and fistulas (Figs. 5–7). Management of acute inflammation of malignant tumors is determined by the clinical picture and the type of neoplasm. Abscesses and fistulas complicating cervical cancer need medical management with antibiotics and percutaneous drainage. In the case of fistulization with the intestines, the role of imaging lies in detecting the level of fistula, which determines the type of surgical management such as defunctioning stoma or resection of the fistula (Fig. 5). Ruptured pyometra is a surgical emergency[21]. Inflamed mucinous neoplasms of the appendix and perforated colonic neoplasms also need prompt surgical attention.
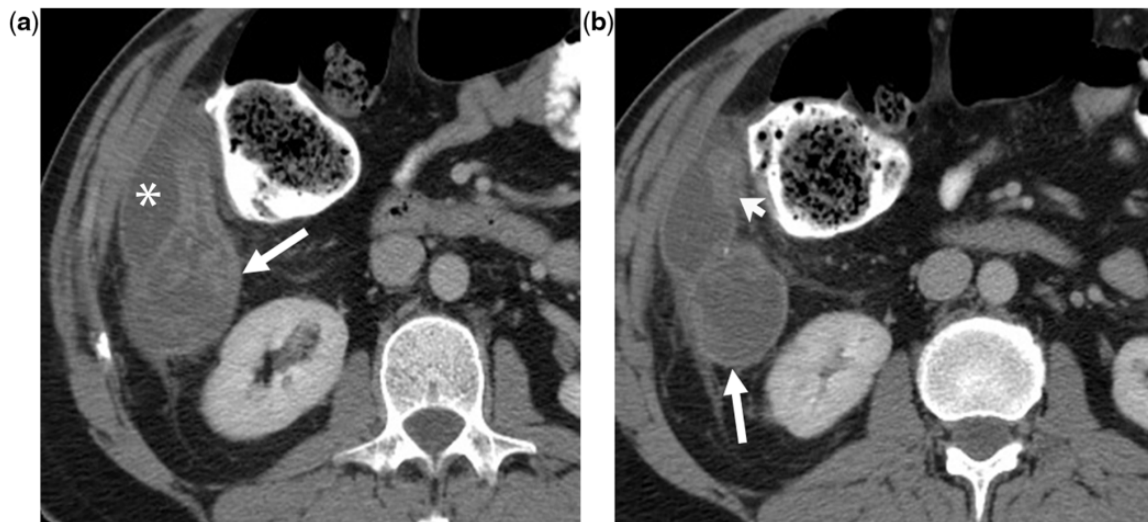
Figure 7 **A 47-year-old man with a ruptured mucinous neoplasm of the appendix. (a,b) Axial contrast-enhanced CT images demonstrate a dilated appendix (arrow) with periappendiceal mucinous material (asterisk) and adjacent soft tissue nodularity (short arrow, b) consistent with a ruptured mucinous neoplasm of the appendix. Histopathology revealed cystadenocarcinoma of the appendix.**

Acute gastrointestinal complications

Malignant bowel obstruction (MBO) can involve the large or small bowel and usually occurs at advanced stages. The incidence of MBO varies between 10% and 28% in colorectal cancer[26]. Clinical criteria for diagnosing MBO include clinical evidence of bowel obstruction (by history, physical or radiologic examination), bowel obstruction beyond the ligament of Treitz, intra-abdominal primary cancer with incurable disease, or non—intra-abdominal primary cancer with clear intraperitoneal disease[27]. The definition excludes MBO occurring proximal to the ligament of Treitz as management in these locations is less controversial with an endoscopic approach established as the primary treatment modality. Management of small and large bowel obstruction, on the contrary, is less standardized. Although extensive surgery is the definitive treatment option in a select group of patients, a substantial number of patients are poor surgical candidates and need less aggressive treatment such as defunctioning colostomy, stenting and medical management of pain[26].

Gastric, pancreaticoduodenal groove and gall bladder cancers may result in malignant gastroduodenal obstruction. Malignant small bowel obstruction is usually secondary to metastatic disease either from diffuse peritoneal carcinomatosis or isolated serosal metastases; primary tumors are extremely rare. The most common extra-abdominal tumors to metastasize to the gastrointestinal tract are melanoma and breast cancer[28]. Up to 2–4% of patients with melanoma develop gastrointestinal tract metastases, which may present as intussusception[29] (Fig. 8). Multiple serosal deposits in the small bowel can result in closed-loop obstruction (Fig. 9).

Malignant large bowel obstruction usually results from primary colorectal cancer, and occasionally from contiguous invasion from tumors of the gall bladder, pancreas, kidney or ovaries[26] (Fig. 10). Sigmoid colon cancers are more often obstructive due to the annular type of growth, solid feces and narrow luminal diameter.

MDCT is the investigation of choice in the management of MBO. Small bowel obstruction due to metastatic deposits is seen on MDCT as dilated small bowel loops with single or multiple transition points, enhancing intraluminal, mural or serosal tumor deposits and focal asymmetric mural thickening[30,31] (Fig. 9). CT findings of malignant intussusception include a bowel-within-bowel configuration with enhancing tumor as the lead point[31] (Fig. 8). Obstruction due to carcinomatosis shows nodular peritoneal thickening and bulky peritoneal masses. MDCT in malignant large bowel obstruction may demonstrate irregular enhancing polypoidal tumor in the colon, pericolonic lymphadenopathy and dilated bowel loops proximal to the tumor.

Bowel ischemia can result from high-grade obstruction, closed-loop type obstruction, bacterial overgrowth or mechanical vascular occlusion. Colonic carcinoma may be complicated by ischemic colitis in 1–7% of cases[32]. Findings suggestive of ischemia include thickened non-enhancing or poorly enhancing bowel loops, submucosal edema, target sign due to alternating layers of enhancement in the bowel wall, pneumatosis intestinalis, gas in the portal vein and direct visualization of occluded mesenteric vessels[33]. Bowel perforation may result from long-standing bowel obstruction or ischemia. Metastatic deposits, especially those from lung cancer, may perforate spontaneously or after chemotherapy[30] (Fig. 11). CT findings in perforation include
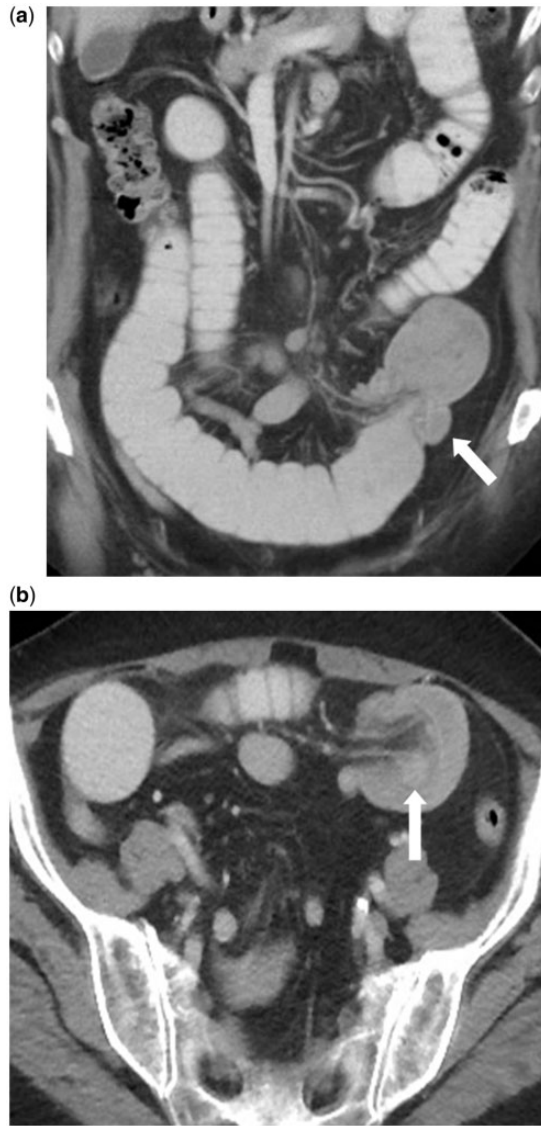
Figure 9 **A 39-year-old man with a past history of small bowel adenocarcinoma, postsurgery, chemotherapy and loop ileostomy, presented with abdominal pain. Coronal reformatted contrast-enhanced CT image demonstrates multilevel intermediate-grade small bowel obstruction caused by serosal metastases (arrows). Note the stomach distended with oral contrast (asterisk).**

Figure 8 **A 56-year-old woman with a previous history of cutaneous melanoma presented with acute abdominal pain and distension. (a) Coronal reformatted contrast-enhanced CT image demonstrates small bowel obstruction secondary to ileo-ileal intussusception (arrow). (b) Axial CT image reveals a small enhancing lesion within the bowel wall which served as the lead point (double-lined arrows). A metastatic deposit from the melanoma was confirmed at surgery and histopathology.**

pneumoperitoneum, abscess or focal collection of air or fluid with phlegmonous changes adjacent to the perforation site and leakage of oral contrast[30] (Fig. 11).

Acute hepatobiliary and pancreatic complications

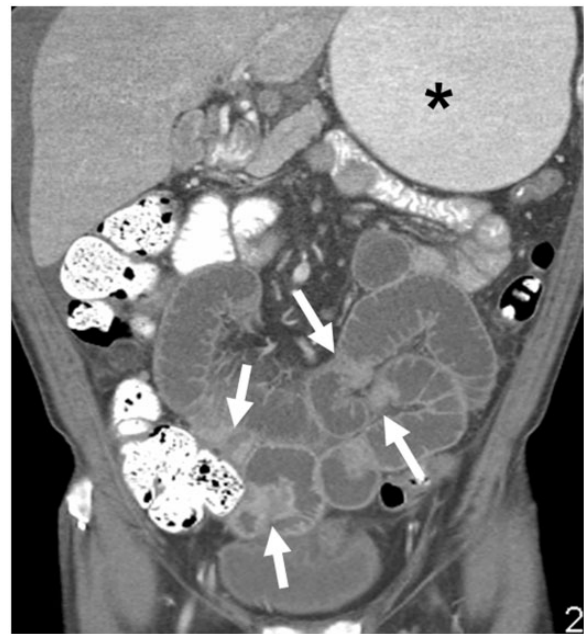Acute liver failure (ALF) refers to acute hepatic dysfunction with hepatic encephalopathy, which occurs within 8 weeks of onset of jaundice in the absence of previous liver disease[34]. In the oncology setting, ALF can occur due to Budd-Chiari syndrome, extensive tumoral hepatic infiltration or severe malignant biliary obstruction[34] (Fig. 12). ALF due to invasion of the hepatic veins or inferior vena cava (IVC) by metastatic lesions or primary malignancies such as IVC leiomyosarcoma, HCC, RCC and adrenal cortical carcinoma (ACC) is rare and is usually associated with a poor prognosis; most patients survive less than 4 weeks[35]. Malignant hepatic infiltration causing ALF is also uncommon and usually seen with hematologic malignancies. Infiltration by solid tumors as a cause of ALF is much less common. Rowbotham et al.[36] in a search of 4020 admissions in a single institute found 18 patients with malignant ALF, of which only 4 patients had solid liver tumors. The most common cause of ALF in this study was non-Hodgkin lymphoma. Clinically, ALF presents as acute abdominal pain, jaundice, hepatic encephalopathy and acute abnormalities in liver function tests. Imaging plays an important role in the workup of patients with ALF and is often requested to exclude obstructive causes of ALF, which can be promptly managed by endoscopic or percutaneous decompression of the biliary system. Ultrasonography is usually the first radiologic investigation performed in these patients. However, MDCT is the definitive investigation as it allows comprehensive
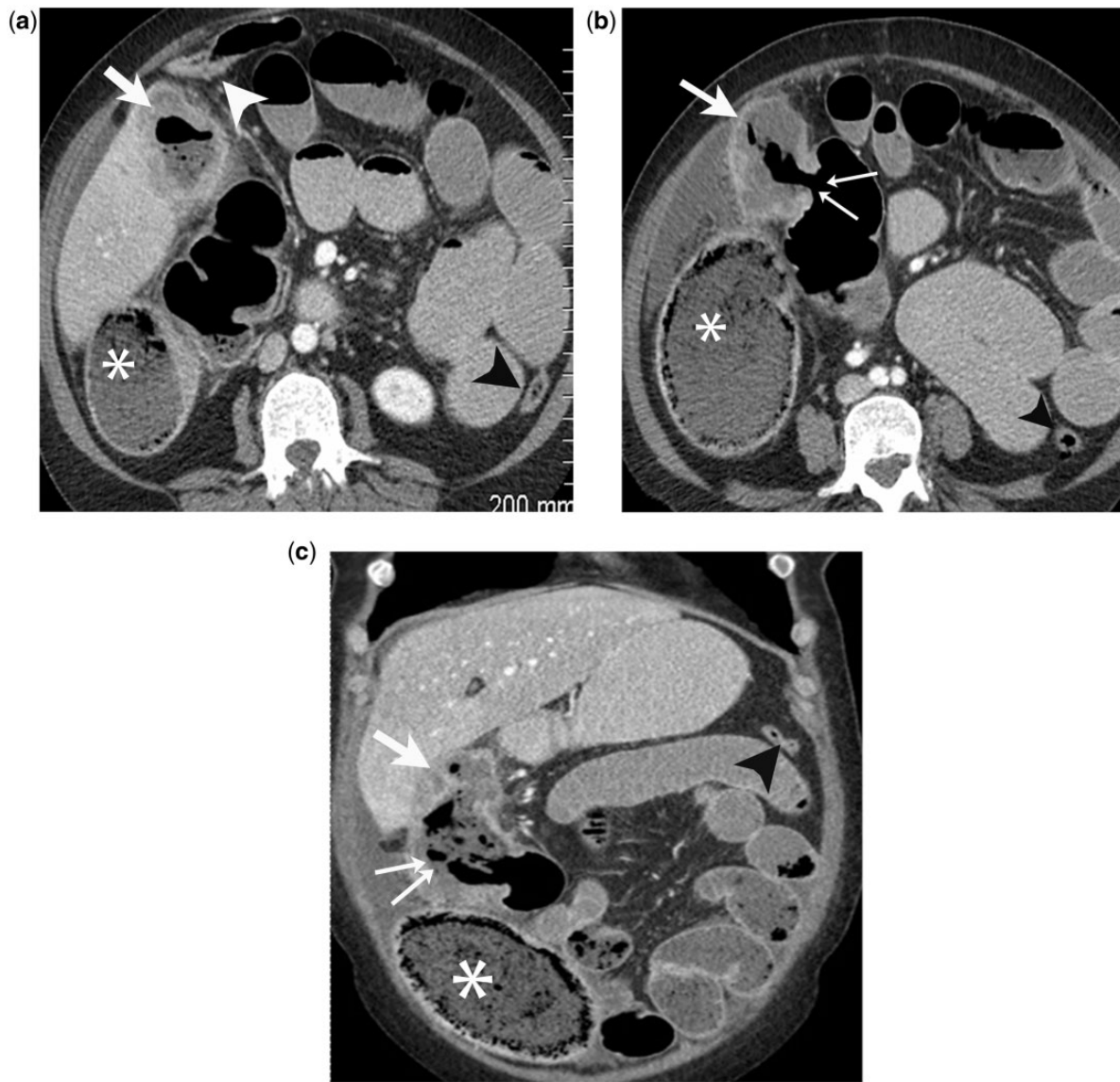
Figure 10 **A 66-year-old woman known to have a gall bladder carcinoma with a cholecystocolic fistula and large bowel obstruction. Plain radiograph of the abdomen revealed small bowel obstruction (not shown). (a,b) Axial and (c) coronal reformatted contrast-enhanced CT images of the abdomen demonstrate irregular thickening of the gall bladder wall and air in the lumen of the gall bladder (thick arrow). There is contiguous invasion of the liver and hepatic flexure of the colon (thick double arrows, c) resulting in a cholecystocolic fistula. There was an associated colonic stricture (white arrowhead) due to tumor invasion resulting in high-grade bowel obstruction. Note the collapsed distal large bowel (black arrowhead). Emergent laparotomy confirmed the cholecystocolic fistula.**

evaluation of both primary and metastatic disease. Findings that may indicate hepatocellular dysfunction as the cause of ALF include the presence of extensive hepatic metastasis, a Budd-Chiari–like picture with tumor thrombus in the IVC and the absence of biliary obstruction (Fig. 12). Management of malignant ALF is difficult. Attempts to recanalize the IVC or hepatic veins in Budd-Chiari syndrome due to malignant causes are usually unsuccessful[37]. Chemotherapy remains the mainstay in the management of ALF due to malignant infiltration.

Acute cholangitis and cholangitic abscesses can complicate malignant biliary strictures due to superimposed infection and can occasionally be the presenting feature. In various studies, malignant biliary obstruction was responsible for 8–19% of cases of cholangitis[38]. In this subset of patients, cholangitis tends to be severe with dismal prognosis compared with benign strictures. Acute suppurative cholangitis is seen on MDCT as thickened enhancing biliary walls with periportal edema. Cholangitic abscesses are seen as hypodense collections with peripheral rim enhancement (Fig. 13). A cluster sign
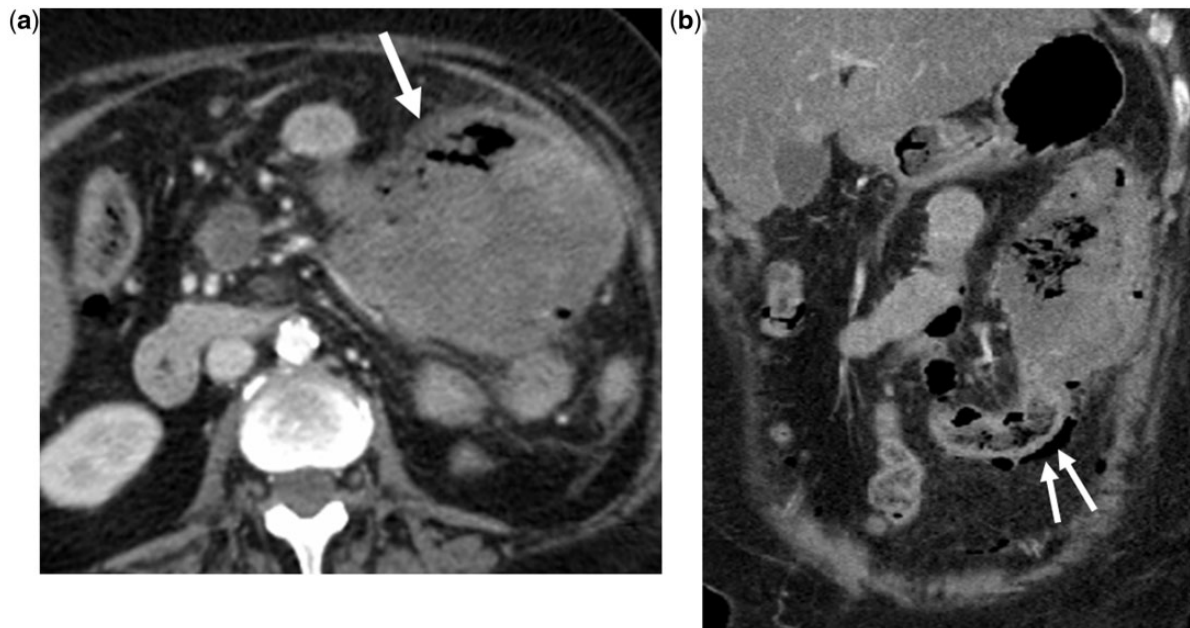
Figure 11 **A 67-year-old woman known to have lung cancer presenting with perforated colonic metastasis. (a) Axial and (b) coronal contrast-enhanced CT images of the abdomen reveal a large heterogeneous necrotic mass (arrow, a) involving the distal transverse colon and splenic flexure. Multiple foci of air are noted in the tumor as well as in the peritoneal cavity (double arrows, b). The patient underwent emergent laparotomy, which revealed perforation of the colonic metastasis.**
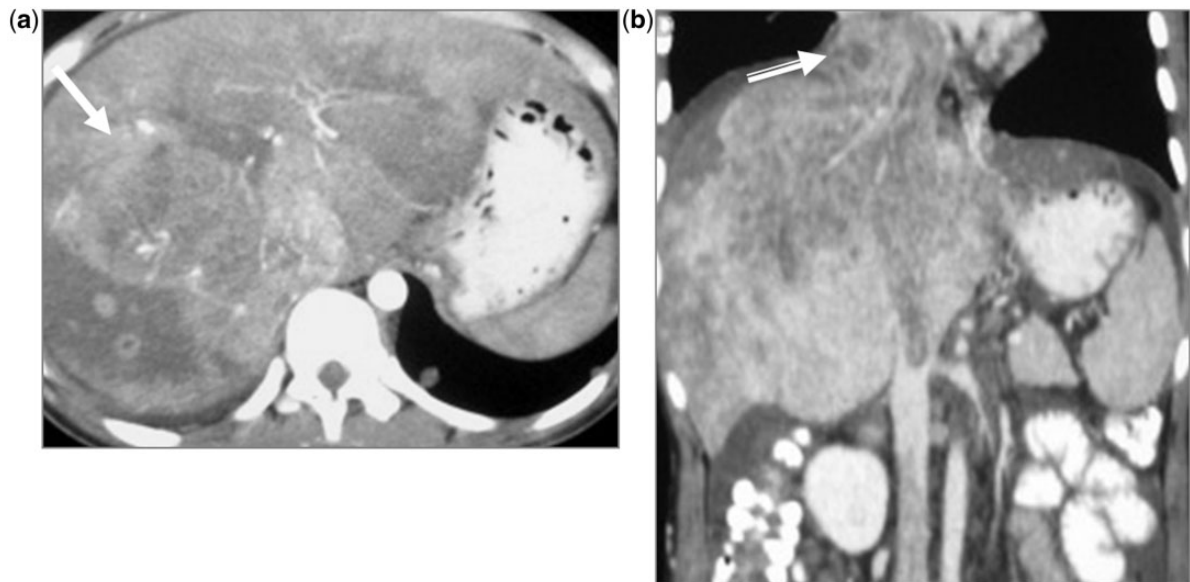

Figure 12 **A 42-year-old man with hepatic lymphoma causing Budd-Chiari syndrome. (a) Axial and (b) coronal reformatted contrast-enhanced CT images of the liver demonstrate a large heterogeneous mass in the liver (arrow, a) with tumor thrombus in the IVC and right atrium (double-lined arrows, b) resulting in Budd-Chiari syndrome. Ultrasound-guided biopsy of the mass revealed a high-grade non-Hodgkin lymphoma.**
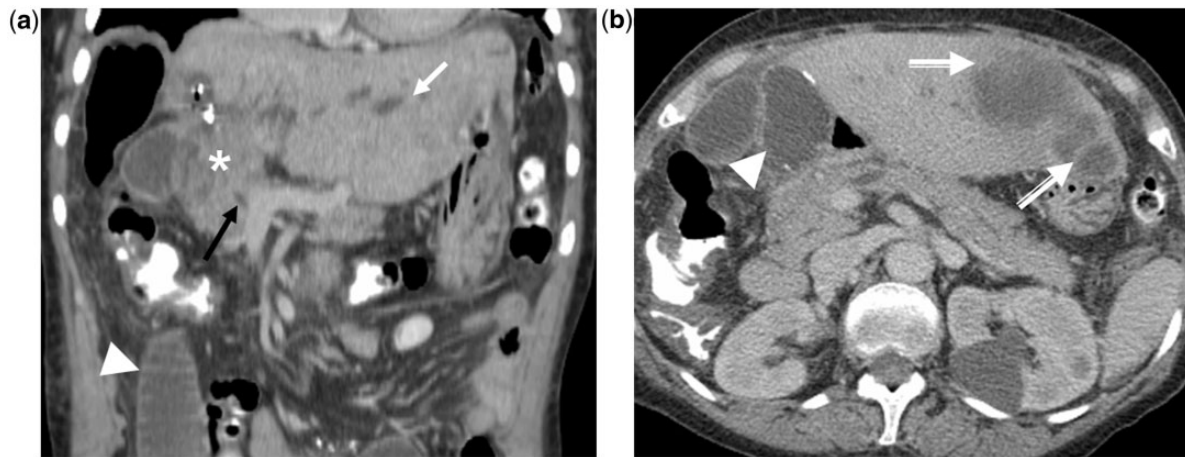
Figure 13 **A 69-year-old woman with a history of extended right hepatectomy, Roux-En-Y hepaticojejunostomy and cholecystectomy for gallbladder carcinoma 1 year previously, now presenting with sepsis. (a) Coronal reformatted and (b) axial contrast-enhanced CT images demonstrate tumor recurrence (asterisk) at the hepatectomy resection margin causing biliary obstruction (white arrow) and portal vein thrombosis (black arrow). The left lateral segment of the liver shows coalescent hypodense lesions (double-lined arrows, b) with irregular walls suggestive of cholangitic abscesses. The Roux-En-Y loop is also obstructed (arrowhead) due to tumor infiltration at one end of the anastamosis and tumor deposit at the other end (not shown). Percutaneous catheter drainage of the abscesses revealed pus with cytology negative for malignancy.**
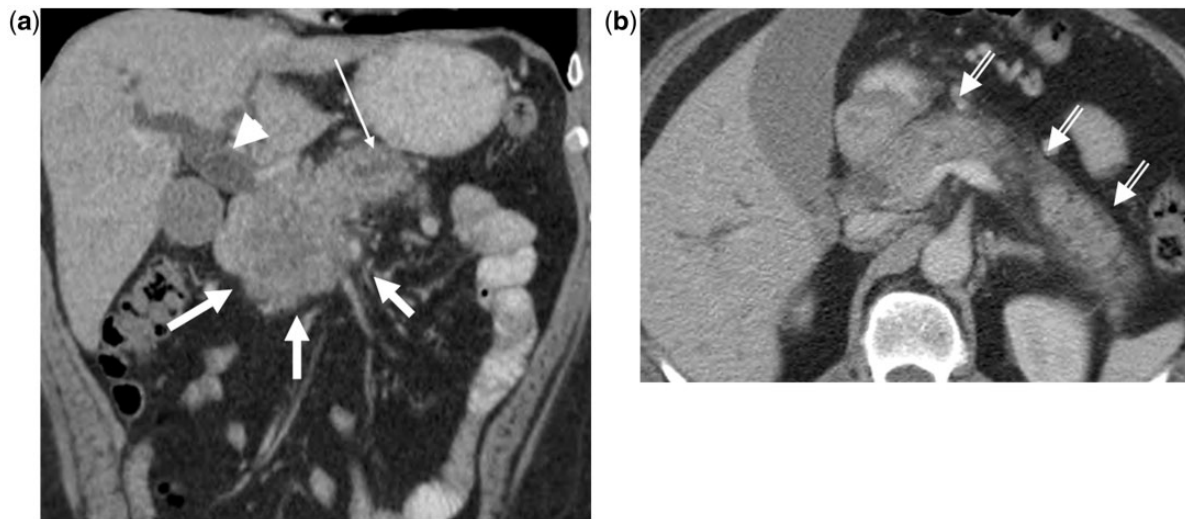

Figure 14 **A 71-year-old man with pancreatic cancer complicated by acute pancreatitis. (a) Coronal reformatted and (b) axial contrast-enhanced CT sections of the abdomen demonstrate a large heterogeneous mass in the pancreatic head (arrows) causing common bile duct (arrow head) and pancreatic duct (thin arrow) obstruction. The body and tail of the pancreas are bulky with peripancreatic fat stranding (double-lined arrows, b) consistent with acute pancreatitis. Serum lipase and amylase levels were acutely increased.**

has been described in cholangitic abscesses, which is secondary to coalescence of multiple abscesses[39]. Acute cholangitis in the setting of malignant biliary obstruction requires endoscopic or nasobiliary drainage with stenting[38]. Cholangitic abscesses need image-guided percutaneous drainage or aspiration.

Acute pancreatitis can complicate primary pancreatic cancer in up to 13% of cases[40] (Fig. 14). Metastatic tumors to the pancreas, most commonly from small cell lung cancer are also known to be complicated by acute pancreatitis[41]. Acute pancreatitis due to pancreatic metastases is encountered in 3.3–7.5% of cases of lung cancer and can occasionally be the presenting feature[41]. Pancreatitis can occur due to malignant hypercalcemia[42]. The MDCT picture of acute pancreatitis is similar to that of other benign causes of pancreatitis and

is seen as pancreatic enlargement with peripancreatic stranding, fluid collection and pancreatic necrosis (Fig. 14). The underlying malignant neoplasm, especially small metastatic deposits, may be obscured by the acute inflammation and may become evident only after the acute inflammation subsides. A careful search for focal pancreatic lesions is therefore important in cancer patients presenting with acute pancreatitis. Acute pancreatitis associated with tumors is managed conservatively.

Acute urinary complications

Acute renal failure (ARF) in the oncology setting can be associated with tumor infiltration. ARF due to involvement of the kidney by solid or hematologic malignancies is uncommon. Although renal involvement is seen in 6–60% of cases of lymphomas and leukemias, the incidence of ARF is less than 0.5%[43]. The most common solid tumors that metastasize to the kidneys are cancers of the lung, stomach and breast[43]. Extensive parenchymal metastasis in these cases can be associated with renal failure. RCC can cause renal failure due to extensive vascular thrombosis. Postrenal causes of ARF can be due to bilateral ureteric obstruction (BUO) or urinary bladder outlet obstruction. BUO resulting in acute reversible kidney injury (ARKI) is a common urologic emergency and is more often due to malignant causes than benign causes. In a study of 49 patients with ARKI, Organ et al.[44] found that 83% cases of BUO were due to malignant causes. The most common malignancies associated with BUO and ARKI are cancers of the cervix, prostate, urinary bladder and colon[44] (Fig. 15). Advanced prostatic cancers can present with acute urinary retention due to bladder outlet obstruction[45]. Imaging helps in the proper triage of oncology patients presenting with ARF by confirming or excluding intrinsic and postrenal causes of ARF. In cases of malignant tumor infiltration, MDCT shows diffuse parenchymal metastasis in the kidneys. The only evidence of lymphomatous infiltration on imaging can be renal enlargement. In postrenal causes of ARF, MDCT helps in detecting the cause and demonstrating the level of obstruction (Fig. 15). ARF due to lymphomatous infiltration responds promptly to systemic chemotherapy. The prognosis for ARF associated with solid tumors depends on the extent of disease elsewhere. The prognosis for ARKI due to BUO in cancer patients is poor; close to 90% patients survive less than 1 year after initial presentation[44]. Management of BUO involves urinary diversion by percutaneous nephrostomy or retrograde ureteric stenting[46] (Fig. 15).

Acute vascular thrombosis

Acute vascular thrombosis with end-organ ischemia is an uncommon presentation of malignant tumors. Vascular
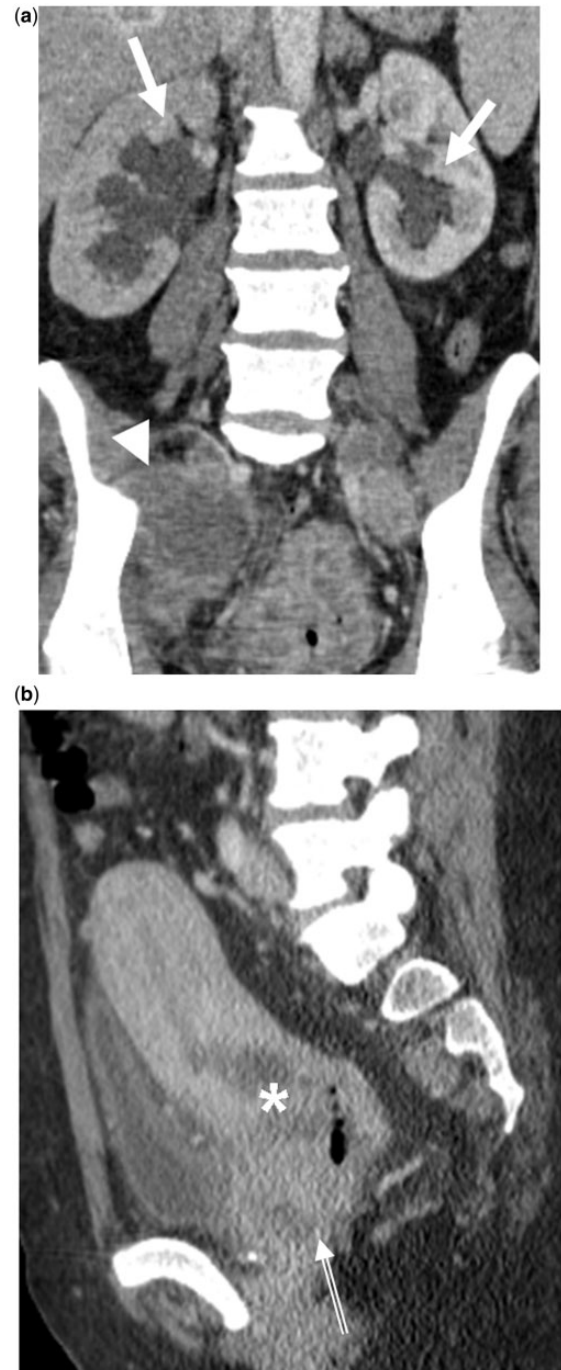

Figure 15 A 43-year-old woman with cervical cancer presented with anuria. (a) Coronal reformatted contrast-enhanced CT image of the abdomen and pelvis reveals bilateral hydronephrosis (arrows) with mild decrease in the enhancement of the right kidney. Note the enlarged necrotic right pelvic side wall lymph nodes (arrowhead). (b) Sagittal reformatted CT image of the pelvis demonstrates a heterogeneous mass in the cervix (asterisk) involving the upper vagina (double-lined arrow). The hydronephrosis was caused by parametrial invasion with extension to the pelvic side wall. Bilateral percutaneous nephrostomies were performed to relieve the urinary obstruction.
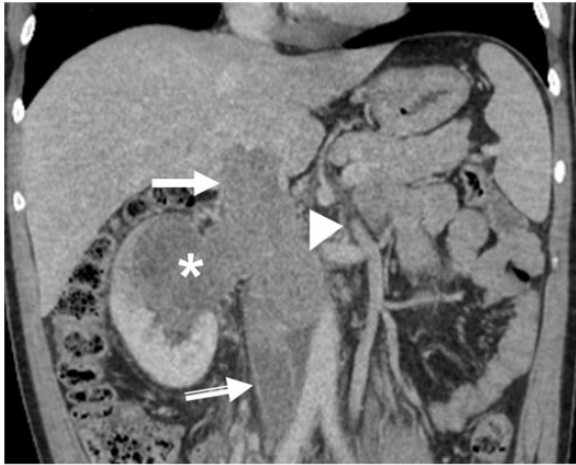
Figure 16 **A 48-year-old man with RCC of the right kidney. Coronal reformatted contrast-enhanced CT image of the abdomen reveals a large right renal mass (asterisk) with contiguous invasion of the right renal vein. The suprarenal IVC is expanded and shows a contiguous enhancing tumor thrombus (arrow). The infrarenal IVC (double-lined arrow) is normal in calibre but shows a bland non-enhancing thrombus due to stasis. Note the enlarged aortocaval lymph node (arrowhead). There were also enlarged paratracheal nodes (not shown) on the CT scan of the chest. Ultrasound-guided biopsy of the right renal mass revealed type 2 papillary RCC. Endobronchial fine-needle aspiration of the paratracheal nodes demonstrated metastasis.**

thrombosis is a common feature of tumors such as HCC, ACC and RCC that contiguously invade the draining veins. Invasion of the IVC is seen in 4–10% cases of RCC[37] (Fig. 16). Malignant portal vein thrombosis is seen in 44% of cases of HCC[47,48]. Despite vascular invasion, infarction of the liver or kidney in HCC or RCC is rare as the vascular invasion develops slowly, allowing the formation of collaterals. In the case of the liver, dual supply from the portal vein and hepatic artery protects the liver from infarction. However, acute thrombosis of the hepatic veins can result in acute Budd-Chiari syndrome due to obstruction of hepatic venous outflow (Fig. 12). Pancreatic carcinoma can invade the superior mesenteric artery and vein, which can result in bowel ischemia[49]. Lymphomatous involvement of the liver can occasionally result in thrombosis of the intrahepatic IVC and Budd-Chiari syndrome (Fig. 12). Metastatic retroperitoneal lymphadenopathy can extend into or compress the IVC with thrombosis of the lower limb veins[50]. MDCT usually demonstrates venous thrombosis as a hypodense intraluminal filling defect with or without luminal dilatation (Fig. 16). Budd-Chiari syndrome is seen on imaging as an enlarged congested liver with decreased enhancement after contrast administration (Fig. 12). Malignant tumor thrombus can be distinguished from bland thrombus based on the presence of

contiguity with the primary tumor, neovascular channels and enhancement of the thrombus[48] (Fig. 16). The upstream veins may show bland thrombosis due to stagnant flow, resulting in acute lower limb deep venous thrombosis. Dislodgement of the thrombus in the lower limbs or in the abdominopelvic veins can result in pulmonary embolism[50]. Management of acute vascular thrombosis revolves around anticoagulation and prevention of pulmonary embolism and end-organ ischemia. Aggressive vascular recanalization may be required in some cases of imminent end-organ ischemia.

Oncologic emergencies in the abdomen and pelvis: role of MDCT

MDCT with oral and intravenous contrast is the most widely used modality in the evaluation of abdominal pain in cancer patients. The key findings that require attention when interpreting the CT images of these patients include changes in the configuration of the tumor, especially changes in the outline, attenuation and enhancement, presence of intratumoral or intra-abdominal high-density fluid, which suggests hemorrhage, abnormal enhancement of blood vessels, solid and hollow viscera, presence of bowel obstruction or urinary obstruction and new sites of disease. Acquisition of unenhanced images may be prudent in patients suspected of intra-abdominal hemorrhage. Comparison with previous imaging can increase the sensitivity of the radiologist in identifying subtle abnormalities.

Conclusion

Acute presentation of abdominal and pelvic malignancies is uncommon but requires prompt detection to decrease morbidity and mortality. MDCT enables accurate detection, localization and characterization of the critical pathologies and permits timely and appropriate management of these patients.

Conflict of interest

The authors have no conflicts of interest to declare.

References

[1] Halfdanarson TR, Hogan WJ, Moynihan TJ. Oncologic emergencies: diagnosis and treatment. Mayo Clin Proc 2006; 81: 835–848. doi:10.4065/81.6.835. PMid:16770986.

[2] Lewis MA, Hendrickson AW, Moynihan TJ. Oncologic emergencies: pathophysiology, presentation, diagnosis, and treatment. CA Cancer J Clin 2011; doi: 10.3322/caac.20124 [Epub ahead of print].

[3] Lai ECH, Lau W. Spontaneous rupture of hepatocellular carcinoma: a systematic review. Arch Surg 2006; 141: 191. doi:10.1001/archsurg.141.2.191. PMid:16490898.

[4] Diaz JR, Agriantonis DJ, Aguila J, Calleros JE, Ayyappan AP. Spontaneous perirenal hemorrhage: what radiologists need to

know. Emerg Radiol 2011; 18: 329–334. doi:10.1007/s10140-011-0944-9. PMid:21344245.

[5] Daskalopoulos G, Karyotis I, Heretis I, Anezinis P, Mavromanolakis E, Delakas D. Spontaneous perirenal hemorrhage: a 10-year experience at our institution. Int Urol Nephrol 2003; 36: 15–19. doi:10.1023/B:UROL.0000032680.65742.9a. PMid:15338665.

[6] Bucher P, Poletti PA, Myit S, Morel P. Spontaneous rupture of a gastrointestinal stromal tumour associated with life-threatening nontraumatic hemoperitoneum. Can J Surg 2008; 51: E38. PMid:18377739.

[7] Casal Rodriguez AX, Sanchez Trigo S, Ferreira Gonzalez L, Brage Gomez S. Hemoperitoneum due to spontaneous rupture of ovarian adenocarcinoma. Emerg Radiol 2011; 18: 267–269. doi:10.1007/s10140-010-0929-0. PMid:21221696.

[8] Park SB, Kim JK, Kim KR, Cho KS. Imaging findings of complications and unusual manifestations of ovarian teratomas1. Radiographics 2008; 28: 969–983. doi:10.1148/rg.284075069. PMid:18635624.

[9] Casillas VJ, Amendola MA, Gascue A, Pinnar N, Levi JU, Perez JM. Imaging of nontraumatic hemorrhagic hepatic lesions1. Radiographics 2000; 20: 367–378. PMid:10715337.

[10] Gulati A, Vyas S, Lal A, et al. Spontaneous rupture of hepatic metastasis from choriocarcinoma: a review of imaging and management. Ann Hepatol 2009; 8: 384–387. PMid:20009141.

[11] Albi G, del Campo L, Tagarro D. Wunderlich's syndrome: causes, diagnosis and radiological management. Clin Radiol 2002; 57: 840–845. PMid:12384111.

[12] Lubner M, Menias C, Rucker C, et al. Blood in the belly: CT findings of hemoperitoneum. Radiographics 2007; 27: 109–125. doi:10.1148/rg.271065042. PMid:17235002.

[13] Furlan A, Fakhran S, Federle MP. Spontaneous abdominal hemorrhage: causes, CT findings, and clinical implications. Am J Roentgenol 2009; 193: 1077–1087. doi:10.2214/AJR.08.2231.

[14] Lai EC, Lau WY. Spontaneous rupture of hepatocellular carcinoma: a systematic review. Arch Surg 2006; 141: 191–198. doi:10.1001/archsurg.141.2.191. PMid:16490898.

[15] Argenta PA, Yeagley TJ, Ott G, Sondheimer SJ. Torsion of the uterine adnexa. Pathologic correlations and current management trends. J Reprod Med 2000; 45: 831–836. PMid:11077633.

[16] Lee R, Welch J. Torsion of the uterine adnexa. Am J Obstet Gynecol 1967; 97: 974. PMid:6021113.

[17] Eitan R, Galoyan N, Zuckerman B, Shaya M, Shen O, Beller U. The risk of malignancy in post-menopausal women presenting with adnexal torsion. Gynecol Oncol 2007; 106: 211–214. doi:10.1016/j.ygyno.2007.03.023. PMid:17482243.

[18] Oltmann SC, Fischer A, Barber R, Huang R, Hicks B, Garcia N. Pediatric ovarian malignancy presenting as ovarian torsion: incidence and relevance. J Pediatr Surg 2010; 45: 135–139. doi:10.1016/j.jpedsurg.2009.10.021. PMid:20105593.

[19] Nakai T, Shimomura T, Nakai H, Suzuki Y, Nakamura M, Kawasaki T. Case report: pedunculated gastrointestinal stromal tumor presenting as bowel volvulus: usefulness of color doppler ultrasonography for strangulated ileus. Digest Dis Sci 2003; 48: 291–294. doi:10.1023/A:1021971209043. PMid:12643604.

[20] Narayanan P, Nobbenhuis M, Reynolds KM, Sahdev A, Reznek RH, Rockall AG. Fistulas in malignant gynecologic disease: etiology, imaging, and management1. Radiographics 2009; 29: 1073–1083. doi:10.1148/rg.294085223. PMid:19605657.

[21] Ou YC, Lan KC, Lin H, Tsai CC, ChangChien CC. Clinical characteristics of perforated pyometra and impending perforation: specific issues in gynecological emergency. J Obstet Gynaecol Res 2010; 36: 661–666. doi:10.1111/j.1447-0756.2010.01184.x. PMid:20598053.

[22] Tirumani SH, Fraser-Hill M, Auer R, Walsh C, Lee F, Ryan J, Shabana W. Mucinous neoplasms of the appendix: A current comprehensive clinico-pathologic and imaging review. Cancer Imaging 2013; 13: 14–25. doi:10.1102/1470-7330.2013.0003. PMid: 23439060.

[23] Lin YT, Liu CJ, Chen TJ, et al. Pyogenic liver abscess as the initial manifestation of underlying hepatocellular carcinoma. Am J Med 2011; 124: 1158–1164. doi:10.1016/j.amjmed.2011.08.012. PMid:22114829.

[24] Devendra K, Tay SK. Metastatic carcinoma of the cervix presenting as a psoas abscess in an HIV-negative woman. Singapore Med J 2003; 44: 302–303. PMid:14560863.

[25] Singh GS, Aikins JK, Deger R, King S, Mikuta JJ. Metastatic cervical cancer and pelvic inflammatory disease in an AIDS patient. Gynecol Oncol 1994; 54: 372–376. doi:10.1006/gyno.1994.1226. PMid:8088616.

[26] Ripamonti CI, Easson AM, Gerdes H. Management of malignant bowel obstruction. Eur J Cancer 2008; 44: 1105–1115. doi:10.1016/j.ejca.2008.02.028. PMid:18359221.

[27] Anthony T, Baron T, Mercadante S, et al. Report of the clinical protocol committee: development of randomized trials for malignant bowel obstruction. J Pain Symptom Manage 2007; 34(1 Suppl): S49–S59. doi:10.1016/j.jpainsymman.2007.04.011. PMid: 17544243.

[28] Krouse RS. The international conference on malignant bowel obstruction: a meeting of the minds to advance palliative care research. J Pain Symptom Manage 2007; 34(1 Suppl): S1–S6. doi:10.1016/j.jpainsymman.2007.04.005. PMid:17544251.

[29] Manola J, Atkins M, Ibrahim J, Kirkwood J. Prognostic factors in metastatic melanoma: a pooled analysis of Eastern Cooperative Oncology Group trials. J Clin Oncol 2000; 18: 3782–3793. PMid:11078491.

[30] Kim SY, Ha HK, Park SW, Kang J, Kim KW, Lee SS, et al. Gastrointestinal metastasis from primary lung cancer: CT findings and clinicopathologic features. Am J Roentgenol 2009; 193: W197–W201. doi:10.2214/AJR.08.1907.

[31] Silva AC, Pimenta M, Guimaraes LS. Small bowel obstruction: what to look for1. Radiographics 2009; 29: 423–439. doi:10.1148/rg.292085514. PMid:19325057.

[32] Rha SE, Ha HK, Lee SH, et al. CT and MR imaging findings of bowel ischemia from various primary causes1. Radiographics 2000; 20: 29–42. PMid:10682769.

[33] Nicolaou S, Kai B, Ho S, Su J, Ahamed K. Imaging of acute small-bowel obstruction. Am J Roentgenol 2005; 185: 1036–1044. doi:10.2214/AJR.04.0815.

[34] Alexopoulou A, Koskinas J, Deutsch M, Delladetsima J, Kountouras D, Dourakis SP. Acute liver failure as the initial manifestation of hepatic infiltration by a solid tumor: report of 5 cases and review of the literature. Tumori 2006; 92: 354. PMid:17036530.

[35] Moreira V, Aller R, De Luis DA, Roman ALS, Ferreiro A. Fulminant acute budd-chiari syndrome stemming from an adrenal tumor. J Clin Gastroenterol 1997; 24: 110–112. doi:10.1097/00004836-199703000-00015. PMid:9077730.

[36] Rowbotham D, Wendon J, Williams R. Acute liver failure secondary to hepatic infiltration: a single centre experience of 18 cases. Gut 1998; 42: 576–580. doi:10.1136/gut.42.4.576. PMid:9616324.

[37] Shih KL, Yen HH, Su WW, Soon MS, Hsia CH, Lin YM. Fulminant Budd-Chiari syndrome caused by renal cell carcinoma with hepatic vein invasion: report of a case. Eur J Gastroenterol Hepatol 2009; 21: 222–224. doi:10.1097/MEG.0b01 3e 32 8305ba06. PMid:19212212.

[38] Kumar R, Sharma BC, Singh J, Sarin SK. Endoscopic biliary drainage for severe acute cholangitis in biliary obstruction as a result of malignant and benign diseases. J Gastroenterol Hepatol 2004; 19: 994–997. doi:10.1111/j.1440-1746.2004.03415.x. PMid:15304115.

[39] O'Connor OJ, O'Neill S, Maher MM. Imaging of biliary tract disease. Am J Roentgenol 2011; 197: W551–W558.

[40] Thomas PC, Nash GF, Aldridge MC. Pancreatic acinar cell carcinoma presenting as acute pancreatitis. HPB (Oxford). 2003; 5: 111–113.

[41] Liu SF, Zhang S, Chen YC, et al. Experience of cancer care for metastasis-induced acute pancreatitis patients with lung cancer. J Thorac Oncol 2009; 4: 1231. doi:10.1097/JTO.0b013e3 181b6b831. PMid:19692933.

[42] Mikata N, Imao S, Tanaka Y, Horiuchi D, Matsumoto G, Hasegawa K. [A case of renal cancer complicated with acute hypercalcemia and acute pancreatitis]. Hinyokika Kiyo 1989; 35: 1907−1910. PMid:2515763.

[43] Humphreys BD, Soiffer RJ, Magee CC. Renal failure associated with cancer and its treatment: an update. J Am Soc Nephrol 2005; 16: 151−161. doi:10.1681/ASN.2004100843. PMid:15574506.

[44] Organ M, Norman RW. Acute reversible kidney injury secondary to bilateral ureteric obstruction. Can Urol Assoc J 2011; 5: 392−396. doi:10.5489/cuaj.11058. PMid: 22154632.

[45] Moul J, Davis R, Vaccaro J, Sihelnik S, Belville W, McLeod D. Acute urinary retention associated with prostatic carcinoma. J Urol 1989; 141: 1375. PMid:2470926.

[46] Nariculam J, Murphy D, Jenner C, et al. Nephrostomy insertion for patients with bilateral ureteric obstruction caused by prostate cancer. Br J Radiol 2009; 82: 571−576. doi:10.1259/bjr/ 38306763. PMid:19153185.

[47] Pirisi M, Avellini C, Fabris C, et al. Portal vein thrombosis in hepatocellular carcinoma: age and sex distribution in an autopsy study. J Cancer Res Clin Oncol 1998; 124: 397−400. doi:10.1007/s004320050189. PMid:9719503.

[48] Willatt JM, Hussain HK, Adusumilli S, Marrero JA. MR imaging of hepatocellular carcinoma in the cirrhotic liver: challenges and controversies1. Radiology 2008; 247: 311−330. doi:10.1148/ radiol.2472061331. PMid:18430871.

[49] Horton KM, Fishman EK. Multi−detector row ct of mesenteric ischemia: can it be done? 1. Radiographics 2001; 21: 1463−1473. PMid:11706217.

[50] Kaufman LB, Yeh BM, Breiman RS, Joe BN, Qayyum A, Coakley FV. Inferior vena cava filling defects on CT and MRI. Am J Roentgenol 2005; 185: 717−726. doi:10.2214/ ajr.185.3.01850717.