

## CHAPTER 16

# Complications of Pelvic Exenteration

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Pelvic exenteration, the en bloc removal of the pelvic organs, is indicated for central recurrent or persistent gynecologic cancer, including cervical, endometrial, vaginal, or vulvar cancer. Even when performed in the setting of specialized centers by highly skilled surgeons, pelvic exenteration is associated with significant morbidity and mortality. Since the initial series published by Brunshwig<sup>1</sup> in 1948, there has been a dramatic change in the type and frequency of the complications associated with this procedure. A number of factors have influenced the outcomes over the past several years, and these include the integration of broad-spectrum antibiotics, thromboembolic prophylaxis, vessel-sealing devices, multiteam surgical expertise, and critical care teams. In addition, modifications of urinary diversion and pelvic reconstruction procedures have paved the way to provide improved outcomes and lower complications rates. Nevertheless, this operation remains a challenge for all patients, and all involved with the care of the patient must recognize that it is a life-changing experience that affects physical, psychological, and sexual function and leads to major changes in quality of life. The report of a series by Maggioni and colleagues<sup>2</sup> showed that the overall morbidity after pelvic exenteration was 66%, with 48% of patients having early complications (<30 days) and 48.5% of patients having late complications. The MD Anderson Cancer Center published a report on 160 patients who underwent pelvic exenteration for gynecologic malignancies and noted that the postoperative complication rate was as high as 94%, with 60% of all complications described as a potentially life-threatening event. The same group also noted a mortality rate of 1.3%.<sup>3</sup> However, such variation may be secondary to the criteria set forth in the respective studies to define complications in the perioperative period. Overall, it is imperative to ensure careful patient selection, preoperative and postoperative care, and optimal surgical expertise in a tertiary cancer center to improve not only surgical outcomes but also survival for patients undergoing this procedure.

This chapter addresses the potential medical and surgical complications that may arise after pelvic exenteration. The emphasis is on the most common signs and symptoms, detection of such complications, and subsequent management options, highlighting surgical versus nonsurgical options. This chapter is intended as a reference guide to aid gynecologic oncologists in assessing the most common complications that arise after pelvic exenteration, and accordingly we do emphasize that appropriate consultation with indicated services is always encouraged.

## Medical Complications

### Febrile Morbidity

One of the most common postoperative complications in patients who have undergone pelvic exenteration is fever. Postoperative fever is defined as a temperature above 38°C (100.4°F) on 2 consecutive postoperative days or above 39°C (102.2°F) on any 1 postoperative day. The differential diagnosis is strongly influenced by the time of onset of the fever. The most common cause of fever within the first 48 hours is a pyretic response to the operation, and this is usually self-limiting. Studies have shown that the rate of febrile morbidity after pelvic exenteration can be as high as 71%.<sup>4</sup> In the study by Westin and colleagues<sup>3</sup> from MD Anderson, the rate of early sepsis (<60 days) was 8.8%, and the rate beyond this time point was 1.3%.

Among the most common causes of fever are the following:

- *Infectious:* Surgical site infection, pneumonia, urinary tract infection, and/or intravascular catheter-related infection
- *Noninfectious:* Hematoma or seroma, deep venous thrombosis (DVT) or pulmonary embolism (PE), inflammatory reaction (pancreatitis), vascular complication (hemorrhage, myocardial infarction, bowel ischemia or infarction), medications

After pelvic exenteration, sepsis may also be a great cause of morbidity and mortality. To be diagnosed with sepsis, a patient must have two of the following signs plus a confirmed infection: body temperature above 38.3°C (101°F) or below 36°C (96.8°F), heart rate higher than 90 beats per minute, and respiratory rate higher than 20 breaths per minute. Severe sepsis is diagnosed when a patient has one of the following: decreased urine output, abrupt changes in mental status, thrombocytopenia, dyspnea, myocardial dysfunction, or abdominal pain.

The routine workup of febrile morbidity should be targeted based on the organ system or infectious process of highest suspicion. The need for laboratory testing should be defined by the findings of a careful history and physical examination. The initial approach to the evaluation should include a complete blood count. Chest x-ray examination, urine cultures, and blood cultures are not indicated for all postoperative patients with fever. One should take into account the timing and the causes of fever. In patients with persistent febrile episodes after pelvic exenteration, one should proceed with abdominal and pelvic computed tomography (CT) scanning to rule out the potential possibility of an intraabdominal abscess.

Treatment for febrile morbidity should be tailored according to the source of the fever. Patients with persistent postoperative fever should be started on broad-spectrum antibiotics after cultures have been obtained. Coverage should be against aerobic gram-negative enteric bacilli and anaerobic organisms. If a source of fever is not apparent and blood cultures show no growth after 48 hours, then discontinuation of antimicrobials should be considered. If the cultures are positive, then antibiotic coverage should be focused on the known causative organism(s). All unnecessary treatments including medications, nasogastric tubes, and intravascular and urinary catheters should be discontinued, when possible, in the febrile patient.

In the setting of sepsis, all patients should be managed with broad-spectrum antibiotics, hemodynamic support such as crystalloids or albumin, vasopressor therapy, blood product administration, and mechanical ventilation, if needed. Discussion of goals of care and prognosis with the patient or family is paramount. Palliative care principles should be considered when appropriate.

## Thromboembolic Events

### Incidence and Guidelines

Among women undergoing major gynecologic surgical procedures without thromboprophylaxis, the risk of DVT ranges from 17% to 40%.<sup>5</sup> This risk is even higher among women undergoing operation for gynecologic cancer. Martino and colleagues<sup>6</sup> estimated the incidence of PE among 507 patients with known or suspected gynecologic cancer undergoing intraabdominal operations and found that the risk of postoperative PE in patients with a diagnosis of cancer was 14 times the risk of postoperative PE in those with benign disease.

Current guidelines for thromboprophylaxis are available from a number of groups, including the American College of Chest Physicians (ACCP),<sup>7</sup> American Society of Clinical Oncology (ASCO),<sup>8</sup> National Comprehensive Cancer Network (NCCN),<sup>9</sup> and American College of Obstetricians and Gynecologists (ACOG).<sup>10</sup> All of the aforementioned guidelines support the recommendation that all patients undergoing abdominal or pelvic surgical procedures for malignancy receive pharmacologic prophylaxis. The ASCO, NCCN, and ACOG guidelines recommend the consideration of continuing prophylaxis for up to 28 days after operation. The recommendation for extended prophylaxis in gynecologic cancer patients is derived from two randomized controlled trials indicating that prolonged thromboprophylaxis reduces the incidence of postoperative venous thromboembolism (VTE). The first study was a double-blind multicenter trial in which patients undergoing planned curative open procedures for abdominal or pelvic cancer received enoxaparin (40 mg subcutaneously) daily for 6 to 10 days. Patients were then randomly assigned to receive either enoxaparin or placebo for another 21 days. The results showed a 60% relative reduction and a 7% absolute reduction in the risk of postoperative VTEs in the prolonged thromboprophylaxis group.<sup>11</sup> In a subsequent study, the investigators evaluated the efficacy and safety of thromboprophylaxis with low-molecular-weight heparin (LMWH) (dalteparin) administered for 28 days versus 7 days after major abdominal surgery for cancer. The results showed that the cumulative incidence of VTEs was reduced from 16.3% among patients receiving short-term thromboprophylaxis to 7.3% among patients receiving prolonged thromboprophylaxis.<sup>12</sup>

In patients undergoing pelvic exenteration, the study by Westin and colleagues showed that the rate of thromboembolic events before 60 days was 1.9% and 5% beyond that time.<sup>3</sup> In a study by Jurado and colleagues,<sup>13</sup> the authors reported a rate of DVT among 45 patients who underwent pelvic exenteration of 11% and a rate of PE of 6.7%. Barakat and colleagues<sup>14</sup> reported a mortality rate of 4.5% from PE after pelvic exenteration. It is interesting to note that in a study by Iglesias and colleagues<sup>15</sup> from MD Anderson Cancer Center, the authors showed that the rate of thromboembolic events was not affected by patient body mass index.

### Signs and Symptoms

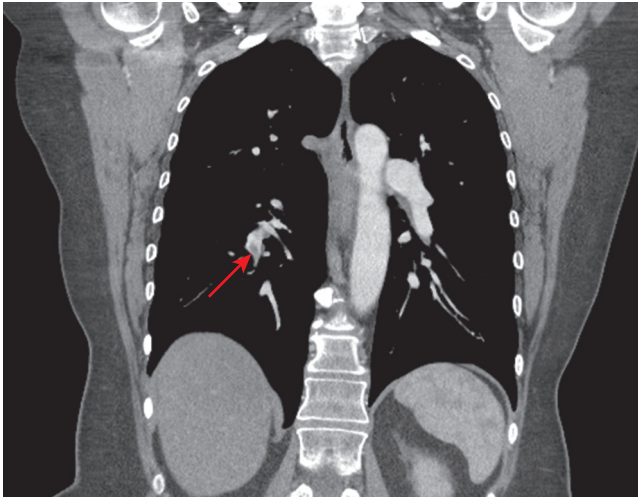
The most common symptoms associated with acute PE include dyspnea (73%), pleuritic chest pain (66%), cough (37%), and hemoptysis (13%). The most common signs are tachypnea (70%), rales (51%), tachycardia (30%), fourth heart sound (24%), accentuated pulmonic component of second heart sound (23%), and circulatory collapse (8%).<sup>16</sup>

### Evaluation of Thromboembolic Events

Once a medical history has been taken and physical examination performed, it is recommended that patients undergo a complete blood count, liver and kidney function tests, and chest radiography and electrocardiography as part of the initial evaluation. In patients with PE, the white blood cell (WBC) count may be normal or elevated, with a WBC count as high as 20,000 K/ $\mu$ L noted in some patients. A chest radiograph may be abnormal in most patients with PE, but the findings are not specific. Common radiographic abnormalities include atelectasis, pleural effusion, parenchymal opacities, and elevation of a hemidiaphragm. It is important to note that a normal-appearing chest radiograph in a patient with severe dyspnea and hypoxemia, but without bronchospasm or cardiac shunt, is strongly suggestive of PE. The most common electrocardiographic abnormalities in the setting of PE are tachycardia and nonspecific ST-T wave abnormalities.

It is important to note that the D-dimer test has limited usefulness in the setting of cancer and thus is not routinely recommended in the workup of thromboembolic events in such patients. Similarly, although arterial blood gas determination may show hypoxemia, hypocapnia, and respiratory alkalosis in patients with a PE, it is not routinely used because of its very low predictive value.

The diagnostic study of choice for DVT is compression ultrasonography. When a DVT is present, the veins do not collapse when pressure is applied. However, it is important to note that a negative ultrasound Doppler result does not rule out DVT, because a number of DVTs may occur in areas that are inaccessible to the ultrasound evaluation. For the diagnosis of PE, the ideal choice of study is computed tomographic pulmonary angiography (Fig. 16.1). This is for patients with a suspected diagnosis of PE and who are hemodynamically stable. However, in patients who are not stable, bedside echocardiography may be used to obtain a presumptive diagnosis to justify the administration of potentially lifesaving therapies. Ventilation-perfusion ( $\dot{V}/\dot{Q}$ ) scanning may be used when CT scanning is not available or if the patient has a contraindication to CT scan or use of intravenous contrast material. Brain natriuretic peptides (BNPs) are neither sensitive (60%) nor specific (62%); however, patients with PE tend to have higher BNP levels. Elevated levels tend to be associated with increased risk of subsequent



**FIG. 16.1** Spiral computed tomography image of the chest with intravenous contrast showing an acute pulmonary embolism (arrow) in the thrombus in the segmental branches of the right lobe of the pulmonary artery.

complications and mortality in patients with PE. BNP testing is not routinely recommended as part of the standard evaluation of PE.

### Treatment of Thromboembolic Events

The approach to a patient with a thromboembolic event is to ensure that the patient's condition has been stabilized after assessment of hemodynamic stability. The first steps should be to provide adequate oxygen supplementation (targeting  $O_2$  saturation  $\geq 90\%$ ), obtain peripheral intravenous access, and begin empiric anticoagulation. The ACCP guidelines recommend starting LMWH or subcutaneous heparin. Once-daily treatment is the preferred choice. The length of anticoagulation for DVT is 3 months, and the recommended length of therapy for PE is 6 months. The ACCP guidelines recommend that thrombolytic therapy should be used in patients with acute PE associated with hypotension (systolic blood pressure BP below 90 mm Hg) who do not have a high risk of bleeding. Embolectomy is recommended in patients with massive PE who have a contraindication to fibrinolysis or who remain unstable after receiving fibrinolysis. It may also be considered in patients with evidence of right ventricular enlargement or dysfunction on transthoracic echocardiogram. Inferior vena cava filters are indicated in the setting of patients with an absolute contraindication to anticoagulant therapy (hemorrhagic stroke or active bleeding). It is also indicated when recurrent embolism is present even after adequate anticoagulant therapy.

### Acute Renal Events

Acute kidney injury (AKI) is the abrupt loss of kidney function, resulting in the retention of urea and other nitrogenous waste products and in the dysregulation of extracellular volume and electrolytes. This term has replaced acute renal failure (ARF) after consideration that even small decrements in kidney function are of substantial clinical relevance and are associated with increased morbidity and mortality. In the study by Westin and colleagues,<sup>3</sup> the authors reported that the rate of ARF or AKI after pelvic exenteration was 3.8%.

**TABLE 16.1** RIFLE Criteria for Acute Renal Compromise<sup>18</sup>

Risk	1.5-fold increase in the serum creatinine, or glomerular filtration rate (GFR) decrease by 25%, or urine output $<0.5$ mL/kg/h for 6 h
Injury	Twofold increase in the serum creatinine, or GFR decrease by 50%, or urine output $<0.5$ mL/kg/h for 12 h
Failure	Threefold increase in the serum creatinine, or GFR decrease by 75%, or urine output of $<0.3$ mL/kg/h for 24 h, or anuria for 12 h
Loss	Complete loss of kidney function (e.g., need for renal replacement therapy) for more than 4 weeks
End-stage renal disease	Complete loss of kidney function (e.g., need for renal replacement therapy) for more than 3 months

AKI has multiple possible causes, and it is most commonly due to acute tubular necrosis (ATN) from ischemia, nephrotoxin exposure, or sepsis. Other frequent causes include volume depletion, urinary obstruction, rapidly progressive glomerulonephritis, and acute interstitial nephritis. AKI is typically detected by means of an increase in serum creatinine and/or a decrease in urine output. Among hospitalized patients, ATN and prerenal disease are the most common causes.

Several consensus definitions of AKI have been developed to provide a uniform definition of AKI. The RIFLE criteria are described here; they consist of three graded levels of kidney dysfunction (risk, injury, and failure), based on the magnitude of increase in serum creatinine or urine output, and two outcome measures (loss and end-stage renal disease [ESRD]). The RIFLE strata are described in Table 16.1.

It has been shown that, compared with patients who did not have AKI, patients in the RIFLE stages of “risk,” “injury,” and “failure” had increased relative mortality risks of 2.4 (confidence interval [CI], 1.94–2.97), 4.15 (CI, 3.14–5.48), and 6.37 (CI, 5.14–7.9), respectively.

### Initial Evaluation After Diagnosis

All patients with AKI must be carefully evaluated both for reversible causes (hypotension, volume depletion, or obstruction) and for the presence of complications (volume overload, hyperkalemia, metabolic acidosis, hypocalcemia, and hyperphosphatemia). The initial evaluation of the patient with AKI is directed at determining the cause and identifying the complications that may require immediate attention. The timing of onset often suggests the underlying cause. A careful review of medications is imperative. Often, nephrotoxic medications have been started before the onset of AKI, which suggests a cause. In addition, even long-standing medications (particularly angiotensin-converting enzyme [ACE] inhibitors or angiotensin receptor blockers) render patients vulnerable to AKI from prerenal factors or ATN.

### Patient Evaluation

The initial assessment should include the careful evaluation of volume status and measurement of serum electrolytes, particularly potassium and bicarbonate, and serum phosphate, calcium, and

albumin. One should also check serum uric acid and magnesium and perform a complete blood count. Initial testing should include reagent strip urinalysis (dipstick) with automated urine microscopy and the quantification of urine protein or albumin (by random or “spot” protein-to-creatinine ratio or albumin-to-creatinine ratio).

A physical examination may reveal the cause. Signs of volume contraction suggest a prerenal cause of AKI. An ultrasound examination could be an option if renal function does not improve; ultrasonography is the most commonly used imaging technique in patients with AKI. Ultrasonography is safe, easy to perform, and sensitive for obstruction. Magnetic resonance imaging (MRI) with gadolinium should be avoided in patients with AKI because of the nephrotoxicity of the agent. In patients with moderate to advanced kidney disease with estimated glomerular filtration rate (eGFR) below 30 mL/min, the administration of gadolinium has been associated with the potentially severe syndrome of nephrogenic systemic fibrosis (NSF).

The results of the urinalysis and ultrasound examination generally direct the remainder of the diagnostic evaluation. Patients who have evidence of obstruction require further investigation and usually intervention to relieve the obstruction and determine the cause. For patients who have normal renal imaging findings, minimal proteinuria, benign urine sediment on urinalysis and microscopy (no red cells or cellular casts), and no clear explanation for AKI, further evaluation is determined by the severity of disease and rate of further decline.

- If the creatinine level is persistently elevated or if an initially mild increase in the creatinine level worsens over the course of days, then a kidney biopsy should be performed. A biopsy is often performed when the diagnosis is uncertain. A biopsy usually enables a more definitive tissue diagnosis and may allow a therapeutic intervention to prevent ESRD.
- In patients who have signs and symptoms of rapidly progressive or unexplained systemic disease, a renal biopsy is warranted, even if the eGFR remains stable after initial increase.
- In patients who have mild decrements in eGFR (e.g., to 45 to 60 mL/min/1.73 m<sup>2</sup>) where the eGFR subsequently remains stable, one should just follow the serum creatinine. If the creatinine level remains stable, one should continue to follow creatinine level, the results of urine studies (urinalysis, microscopic studies, urine protein and creatinine), and blood pressure until a clear temporal pattern has been established.

### Urinalysis

The urinalysis involves both use of a urine dipstick and microscopic examination of the urine sediment. The dipstick can be used to test for protein (albumin), pH, glucose, hemoglobin (or myoglobin), leukocyte esterase (reflecting pyuria), and specific gravity.

### Urine Sodium Excretion

The fractional excretion of sodium (FENa) measures the percent of filtered sodium that is excreted in the urine.

- The FENa is commonly used to assist in differentiating prerenal disease (a reduction in glomerular filtration rate [GFR] due solely to decreased renal perfusion) from ATN, the two most common causes of AKI.
- In patients with suspected prerenal disease or ATN, it is recommended that the FENa be measured. A value of the FENa below 1% commonly indicates prerenal disease; in comparison, a value between 1% and 2% may be seen with either disorders, and a value above 2% usually indicates ATN.

### Urine Volume

Trends in and comparisons between the volumes of fluid going into and coming out of a patient (including urine output) are helpful physiologic parameters in patients with AKI. Oliguria (typically defined as <0.3 mL/kg/h or <500 mL/day of urine output) may or may not occur in patients with AKI. Normal urine output can be maintained even with an abnormally low GFR in patients with nonoliguric ATN. The prognosis of nonoliguric AKI is generally better than that of oliguric or anuric disease.<sup>19,20</sup>

### Management

#### Volume Issues

An assessment of volume status is performed in all patients with AKI because correction of volume depletion or volume overload (especially when associated with worsening cardiac output) may reverse or ameliorate AKI.

#### Volume Depletion

Unless contraindicated, the patient with a clinical history consistent with fluid loss (such as vomiting and diarrhea), physical examination findings consistent with hypovolemia (hypotension and tachycardia), and/or oliguria should receive intravenous fluid therapy. This fluid challenge attempts to identify prerenal failure that can progress to AKI if not treated promptly. Studies have shown that prompt reversal of volume depletion may prevent or limit kidney injury due to ATN. However, such fluid infusion is contraindicated in those with obvious volume overload or heart failure. Fluids may be either crystalloid or colloid. One should begin with 1 to 3 L of fluid, with careful and repeated clinical assessment to evaluate the patient's response to this therapy. Fluid therapy should be targeted to physiologic end points.

#### Volume Overload

Hypervolemia may be present at initial evaluation or may occur because of excessive fluid administration in the setting of impaired ability to excrete sodium and water. This is especially true for patients with sepsis, who commonly receive aggressive intravenous fluid resuscitation.

#### Hyperkalemia

Hyperkalemia is a common and potentially life-threatening complication of AKI. In general, all patients with AKI and hyperkalemia that is refractory to medical therapy should be dialyzed unless hyperkalemia is mild (i.e., <5.5 mEq/L) and the cause of AKI is known to be easily reversed (such as prerenal AKI due to volume depletion or ACE inhibitors).

### Prognosis

Most patients with AKI recover renal function, with recovery manifesting with an increase in urine output and a gradual decrease in the blood urea nitrogen (BUN) and serum creatinine concentration. However, in many patients, including those with previously normal renal function, renal function does not return to baseline levels. In addition, many studies have demonstrated an increase in the risk of chronic kidney disease (CKD) and ESRD in patients who recover from AKI. Even small, acute rises in serum creatinine as low as 0.3 mg/dL (27 μmol/L) are associated with both short-term and long-term increases in mortality.

## Surgical Complications

### Wound-Related Events

#### Superficial Wound Separation

A frequent complication in patients who undergo pelvic exenteration is wound complication. The rate of such complications has ranged from 5.6% to 29.4% in the largest published series. However, one must consider that these include both abdominal wound complications and perineal wound issues.<sup>2-4</sup>

#### Dehiscence and Evisceration

Complete fascial dehiscence is associated with a mortality rate of 10%. Early postoperative fascial dehiscence is a surgical emergency and should be addressed promptly. The risk factors for fascial disruption are advanced age, chronic pulmonary disease, anemia, postoperative coughing, wound infection, and complexity of surgery. Other factors include malignancy, obesity, sepsis, hypoalbuminemia or poor nutrition, and chronic glucocorticoid therapy. Herniation is more common when the incision length exceeds 18 cm.<sup>21</sup> Dehiscence is most likely due to placement of the suture too close to the edge or under tension. To minimize this complication, elective midline abdominal closure should be performed with continuous absorbable sutures.

Signs and symptoms of complete dehiscence include profuse serosanguineous drainage, fever, and abdominal pain. Most dehiscences occur 4 to 14 days after operation. The diagnosis is made primarily based on clinical suspicion. Ultrasonography or CT may be used when the diagnosis is not clear (Fig. 16.2). Once the diagnosis has been confirmed, one should place a moist dressing over the wound at the bedside. When the patient is taken to surgery, the surgeon should perform complete wound opening and subsequent debridement of the fascial edges while ensuring that no bowel injury occurs during the procedure. A mass closure with continuous delayed absorbable sutures should be performed. However, if the fascial defect that remains after proper debridement is too large, use of a wound mesh should be considered.

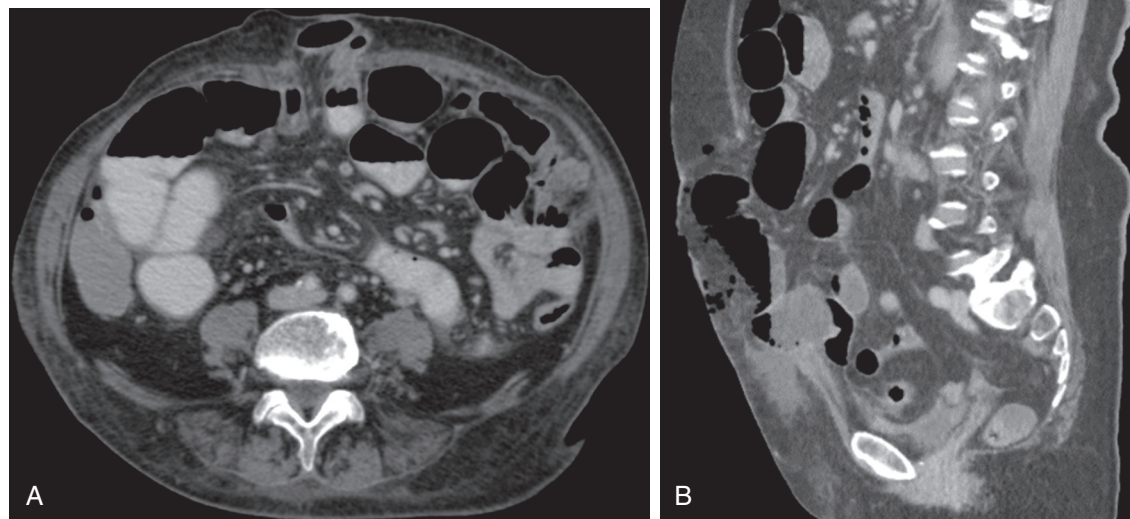
#### Necrotizing Fasciitis

Necrotizing fasciitis is a rare, life-threatening soft tissue infection primarily involving the fascia and subcutaneous tissue. The reported mortality of necrotizing fasciitis ranges from 20% to 80%.<sup>22</sup> There are three types of necrotizing fasciitis. These are:

- Type I—This is a mixed infection caused by both anaerobic and aerobic species. Risk factors include diabetes, peripheral vascular disease, immune compromise, or recent operation.
- Type II—This is generally a monomicrobial infection caused by group A streptococci or other  $\beta$ -hemolytic streptococci, either alone or in combination with other species, most commonly *Staphylococcus aureus*.
- Type III—This is also known as “gas gangrene” and is caused by the organism *Clostridium perfringens*.

Clinical symptoms include erythema, swelling, changes in skin coloring, intense pain that may be disproportionate to the skin findings, subcutaneous emphysema, fever, nausea, vomiting, and/or malaise. It may often be misdiagnosed as cellulitis or abscess. On physical examination, the patient may appear deceptively well; however, this may significantly delay the diagnosis. Such delay will lead to a rapid deterioration of the patient's condition, and the patient will suddenly demonstrate a toxic appearance. The redness quickly spreads, with the margins moving rapidly into normal skin near the site of the incision. The skin will then develop a dusky or purplish discoloration, subsequently leading to large areas of gangrenous skin. Ultimately, anesthesia in the involved region may be reflective of the fact that there is thrombosis to the subcutaneous blood vessels that leads to necrosis of the nerve fibers. Local crepitation can occur; however, this is not a common finding.

Necrotizing fasciitis remains a clinical diagnosis. Imaging studies may be useful to determine whether muscle tissue is involved but should not delay surgical intervention. Early



**FIG. 16.2** Computed tomography scan of abdomen and pelvis with contrast ([A] axial and [B] sagittal planes) showing anterior abdominal fascial dehiscence with evisceration.

radiographic findings include soft tissue thickness and opacity. CT scans may show dermal thickening, increased soft tissue attenuation, inflammatory fat stranding, and possible superficial or deep crescentic fluid or air in the subfascial planes.<sup>23</sup>

The treatment of patients with necrotizing fasciitis consists of adequate and aggressive surgical debridement, supportive care, and broad-spectrum antibiotics. Surgical exploration is the only way to definitely establish the diagnosis. At operation the findings will reveal that the normal skin and subcutaneous tissue become loosened from the rapidly spreading deep necrotic fascia. It is important to note that fascial necrosis is usually more advanced than the appearance suggests. Without prompt treatment, secondary involvement of the deeper muscle layers may occur, resulting in myositis or myonecrosis. It is important to recognize that surgical debridement is associated with a lower mortality when performed within 24 hours of diagnosis.<sup>24</sup>

### Urinary Diversion Complications

During a total or anterior pelvic exenteration, urinary diversion is routinely performed. Patients can choose either continent or incontinent urinary diversion, and there are advantages and disadvantages associated with both of these techniques. Incontinent diversion is faster and less technically challenging than continent diversion; also, incontinent diversion may have the advantage of requiring less maintenance effort and self-care by the patient. The incidences of early and late complications of incontinent urinary diversion have been reported to be 33% and 28%, respectively.<sup>25</sup> The most commonly reported complications are anastomotic leakage (3%), fistula formation (3%–19%), need for reoperation (8%–19%), renal insufficiency (6%–17%), urostomy stricture (7%), and ureteral obstruction (7%) (Fig. 16.3).

Continent urinary diversion offers better cosmetic results than incontinent diversion; however, overall complication rates with continent diversion remain significant and range from 37% to 66%. The most common complications associated with continent urinary diversion are pyelonephritis (13%–42%), difficulty with catheterization (12%–54%), ureteral (anastomotic) stricture (2%–22%), urostomy stricture (4%–22%), incontinence (7%–13.3%), urinary stone formation (7%–18%) (Fig. 16.4), ureteral (anastomotic) leaks (2%–14%), fistula (2%–15%), and permanent renal failure (3%). There is also the potential risk of development of hyperchloremic metabolic acidosis.

In a study by Ramirez and colleagues<sup>26</sup> from MD Anderson Cancer Center, the authors reported on 133 patients who underwent total pelvic exenteration. Ninety-nine patients (74.4%) underwent a total pelvic exenteration, and 34 (25.6%) underwent an anterior pelvic exenteration. In 46 patients (34.6%), continent urinary diversion was performed, and incontinent urinary diversion was performed in 87 patients (65.4%). The mean age at exenteration was 47.6 years (range, 30–73 years) in the continent urinary diversion group and 57.2 years (range, 27–86 years) in the incontinent urinary diversion group ( $P < .0001$ ). Median follow-up time after exenteration was 28.5 months (range, 2.3–185.7 months) for patients with continent urinary diversion and 28.1 months (range, 1.4–187.1 months) for patients with incontinent urinary diversion. The most common postoperative complication was pyelonephritis or urosepsis, which occurred in 32.6% of the patients with continent urinary diversion and 37.9% of the patients with incontinent urinary diversion ( $P = .58$ ). The second most common complication was urinary stone formation, which occurred in 34.8% of the patients with continent urinary diversion and 2.3% of the patients with incontinent urinary diversion

( $P = .001$ ). No stone formation was observed in the first 60 days after continent urinary diversion. Of the 16 patients with stone formation and continent urinary diversion, 11 were asymptomatic and did not require intervention. Three patients underwent laparotomy for stone removal—one because of an enterocutaneous (pouch-to-skin) fistula, possibly secondary to infection and an obstructive mucous plug, and the other two because of large size (one patient) and number of stones ( $n = 1$ ). One patient had bilateral nephrostomy tubes placed because of urinary obstruction and poor functional status, and one patient was treated successfully with cystolitholapaxy. Both patients with stone formation and incontinent urinary diversion were asymptomatic and did not require intervention.

No significant differences were observed between the continent and incontinent urinary diversion groups for rates of ureteral (anastomotic) leakage, ureteral (anastomotic) stricture, renal insufficiency, fistula formation, conduit reoperation, or pyelonephritis or urosepsis. No statistical significance in urostomy stricture formation was found after multivariate analysis ( $P = .08$ ). In patients with at least one episode of pyelonephritis or urosepsis, there was no significant difference between the groups ( $P = .20$ ). There was also no significant difference between the groups for the number of hospitalizations required because of complications related to the urinary diversion ( $P = .45$ ). When the analysis was limited to patients who had received preoperative pelvic radiation, there was an increased incidence of urostomy stricture after 60 days in patients with continent urinary diversion on univariate analysis. Of the patients with continent urinary diversion, 28.3% reported incontinence, and 15.2% reported difficulty with catheterization.

In that study, the authors concluded that patients undergoing pelvic exenteration have a high risk of complications and that there is no difference in postoperative complication rates related to urinary diversion except that urinary stone formation is more common among patients with a continent urinary diversion. Continent urinary diversion is also associated with the potential for additional complications: incontinence and difficulty with catheterization.

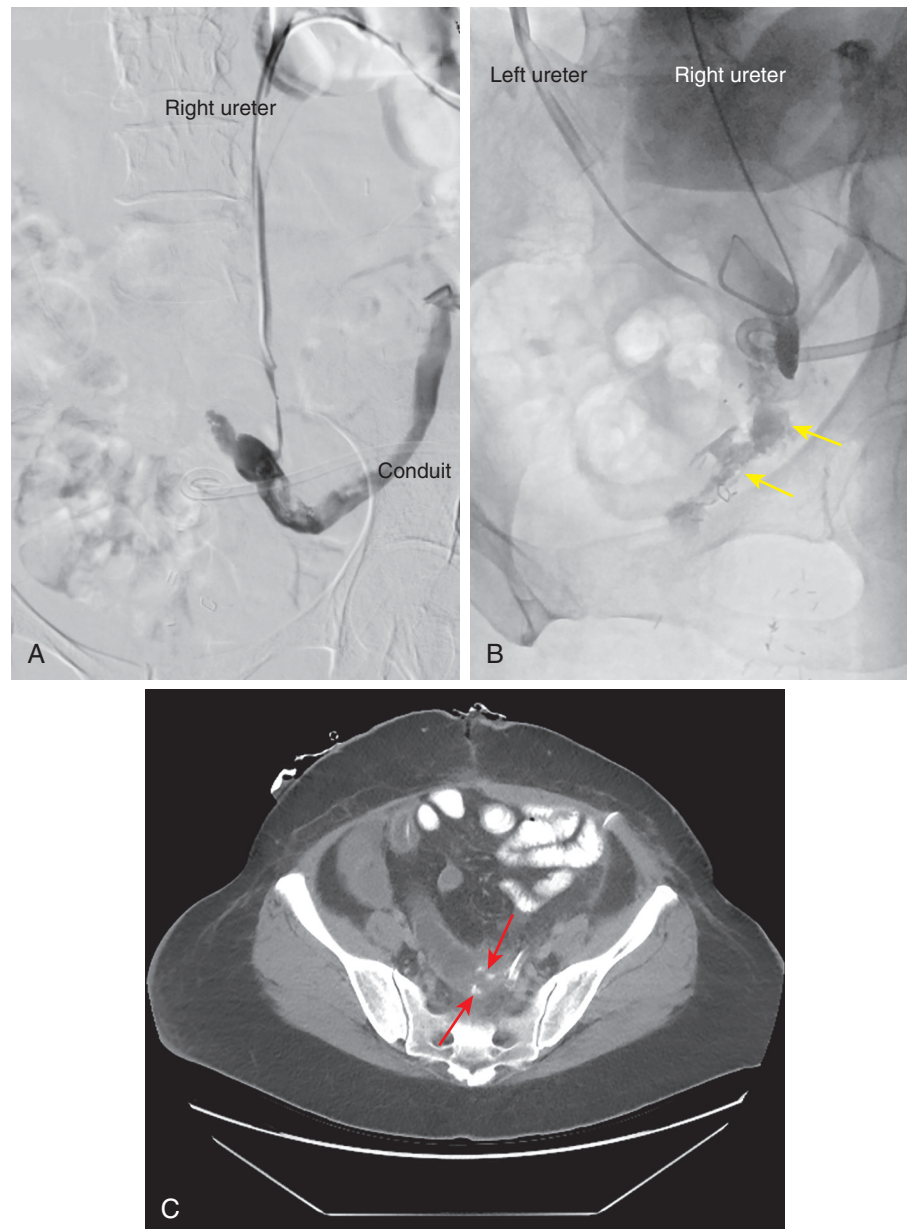
### Bowel-Related Complications

Patients undergoing a pelvic exenteration are at a higher risk of developing postoperative bowel complications as a result of various factors that affect healing, such as poor nutritional status and prior history of radiation therapy. It has been shown that the rate of bowel-related complications after pelvic exenteration is approximately 10%.<sup>3,4</sup>

#### Postoperative Ileus

Postoperative paralytic ileus refers to obstipation and intolerance of oral intake due to nonmechanical factors that disrupt the normal coordinated propulsive motor activity of the gastrointestinal tract following abdominal or nonabdominal surgical procedures. After abdominal operation, “normal” physiologic postoperative ileus due to postoperative gut dysmotility is widely reported as lasting 0 to 24 hours in the small intestine, 24 to 48 hours in the stomach, and 48 to 72 hours in the colon.<sup>27</sup> The multiple definitions of “prolonged” postoperative ileus have included:

- No return of bowel function postoperatively (ranging from postoperative days 4 to 6)
- Absence of flatus or stool by postoperative day 6



**FIG. 16.3** (A) Digital subtraction angiogram showing filling of the conduit without evidence of leakage of the right ureteral anastomosis (normal right ureter drainage). (B) Left posterior oblique view demonstrating contrast leaking from left ureter into the pelvis (*arrows*). (C) Computed tomography scan of the abdomen and pelvis with contrast. Urinary leak at ureteric anastomosis with the ileal conduit in the posterior left pelvis.

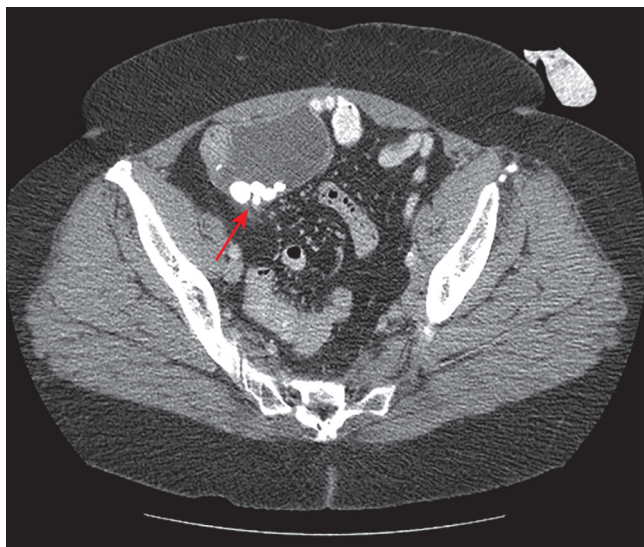
- Postoperative nausea or vomiting necessitating cessation of oral intake, intravenous support, or nasogastric tube placement by postoperative day 5
- Return of bowel function after postoperative day 5
- Absence of flatus and/or bowel movement prolonging hospitalization beyond discharge goal (ranging from postoperative days 6 to 8)
- Lack of bowel activity more than 5 days after operation

Among the most common nonsurgical risk factors are opioid use, antihypertensive agents, antidiarrheal or antiemetic agents, any drug with an anticholinergic property, muscle relaxants, and atropine products. There are also a number of medical conditions that may predispose the patient to postoperative ileus. These include pancreatitis, gastroenteritis, spinal cord injury, myocardial infarction, stroke,

pneumonia, diabetes, diabetic ketoacidosis, botulism, or Parkinson disease. When considering surgical factors, one must consider that lower abdominal procedures with large incisions and with intestinal manipulation (e.g., colorectal, gynecologic [exenteration]) are associated with a higher risk of postoperative ileus, whereas abdominal procedures with smaller incisions and minimal visceral manipulation (e.g., cholecystectomy) are associated with a lower risk.

The most common symptoms are abdominal distention, bloating, diffuse abdominal pain, nausea and/or vomiting, inability to pass flatus, and inability to tolerate a regular oral diet. On examination, the patient may have abdominal distention and a tympanic abdomen with reduced bowel sounds and some degree of tenderness.

The diagnosis is established based on clinical findings and plain abdominal films. These may show dilated loops of bowel



**FIG. 16.4** Computed tomography scan of the abdomen and pelvis with contrast. Multiple urinary continent conduit calculi. Arrow demonstrates site of stones.

but with evidence of air in the colon and rectum without a transition zone that would suggest bowel obstruction (Fig. 16.5A). There should also not be any evidence of free air that is associated with perforation. The diagnosis is established when the signs and symptoms persist for more than 3 to 5 days. When in doubt, CT of the abdomen will help differentiate small bowel obstruction from ileus, given that it has a sensitivity and specificity of 90% to 100%.

Therapy for patients with postoperative ileus should focus on removal of any recognized inciting factors, maintenance and replacement of fluid therapy, electrolyte replacement, bowel rest and bowel decompression (as needed), and serial abdominal examinations.

### Bowel Obstruction

Small bowel obstruction can be functional or mechanical. The small bowel is involved in about 80% of cases of mechanical bowel obstruction.<sup>28</sup> There are several causes for small bowel obstruction; however, in the postoperative period after pelvic exenteration, the most common cause of small bowel obstruction is adhesion formation. It is imperative to diagnose the bowel obstruction early so that the appropriate management may be initiated. In simple mechanical obstruction, blockage occurs without vascular compromise. The normal secretory and absorptive functions of the mucosa are depressed, and the bowel wall becomes edematous and congested. There may also be transudative loss of fluid from the intestinal lumen into the peritoneal cavity. Electrolyte loss is common in this setting, leading to metabolic alkalosis, and the fluid loss may result in hypovolemia. If the bowel obstruction is not recognized and properly addressed, the obstruction will lead to vascular compromise, and the blood flow to the bowel will diminish. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcts, leading to gangrene and perforation. Acute mechanical small bowel obstruction is a common surgical emergency.

### Signs and Symptoms

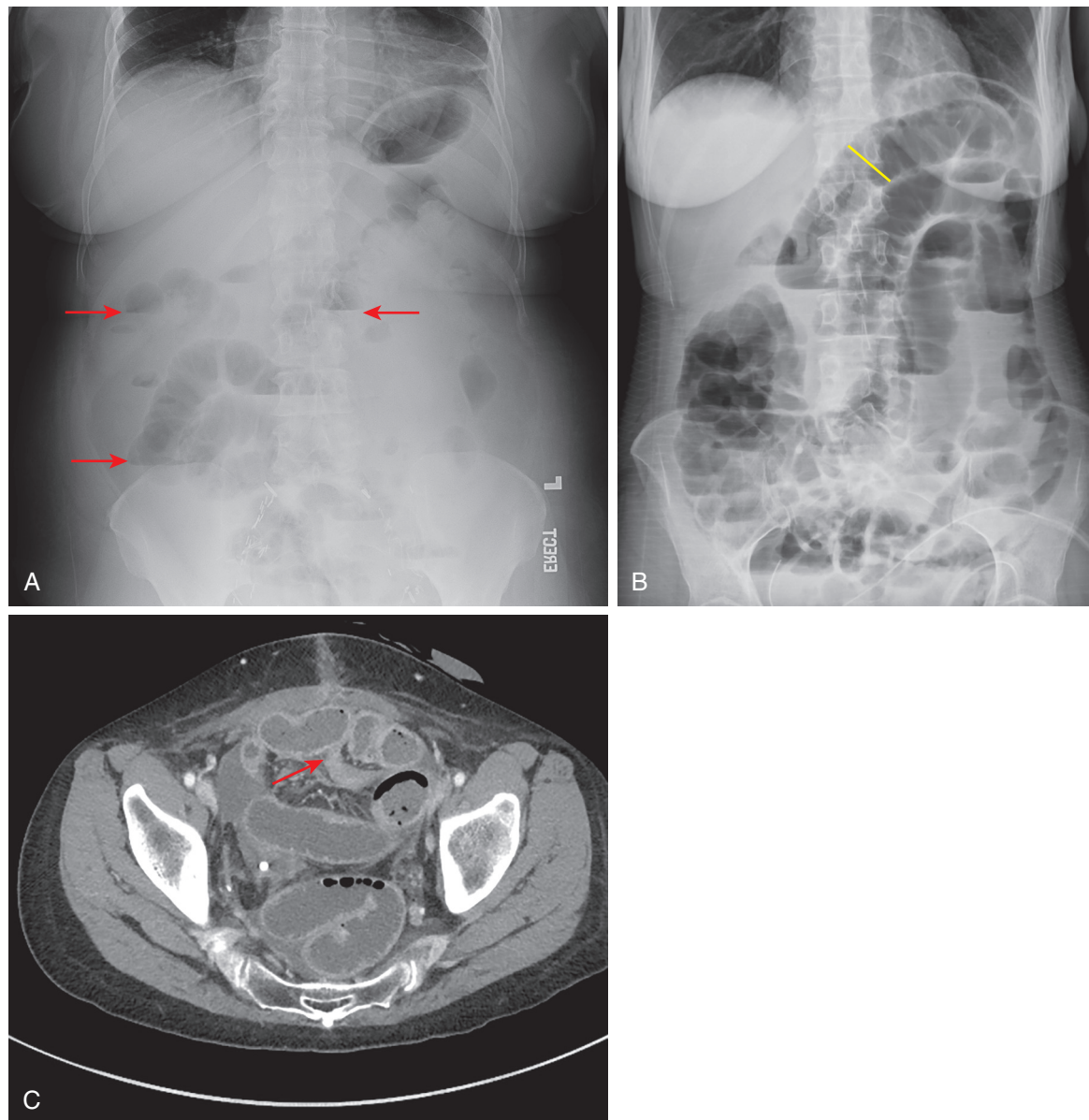
Patients with bowel obstruction may have an abrupt onset of abdominal pain, nausea, vomiting, cramping, and abdominal distention. Patients with partial obstruction may have intermittent episodes of diarrhea; however, more commonly, patients with complete obstruction will have obstipation at presentation. One should note that the presence of diarrhea in the setting of bowel obstruction does not automatically indicate resolution of the obstruction. At inspection of the abdomen, the physical examination may reveal evidence of distention, hyperactive bowel sounds secondary to high-pitched peristalsis, and tenderness. In multiple retrospective reviews, abdominal distention was the most frequent physical finding on clinical examination, occurring in 56% to 65% of patients.<sup>29,30</sup> With significant bowel distention, bowel sounds may become muffled, and as the bowel distention progresses, the bowel sounds may become hypoactive. The degree of tenderness is dependent on the level of obstruction and whether there is evidence of bowel ischemia. Fever may be associated with complications of obstruction such as ischemia or necrosis.

### Diagnosis

In general, the diagnosis of small bowel obstruction is a clinical and radiologic diagnosis. The initial evaluation should include supine and upright abdominal radiographs. X-ray findings are diagnostic in 50% to 60% of patients; equivocal in about 20% to 30%; and normal, nonspecific, or misleading in 10% to 20%.<sup>31</sup> The key radiographic signs that allow distinction between a high-grade small bowel obstruction and a low-grade obstruction are the presence of small bowel distention, with maximal dilated loops averaging 36 mm in diameter and exceeding 50% of the caliber of the largest visible colon loop, in addition to a 2.5-times increase in the number of distended loops in the abdomen compared with the normal number. Other findings that are most significant and predictive of high-grade small bowel obstruction are the presence of more than two air-fluid levels, air-fluid levels wider than 2.5 cm, and air-fluid levels differing more than 2 cm in height from one another within the same small bowel loop.<sup>32</sup> It is often difficult to differentiate postoperative ileus from an obstruction based solely on findings on abdominal radiographs. In that setting, a definitive diagnosis is attained based on both clinical suspicion and CT scan findings (see Fig. 16.5B and C). It should be noted that in patients with necrosis or gangrene, the abdominal imaging may demonstrate gas in the bowel wall, also known as pneumatosis intestinalis (Fig. 16.6). This is an ominous sign and a surgical emergency because imminent bowel perforation is usually seen in this setting.

Standard CT is the ideal imaging modality for evaluation of small bowel obstruction, with sensitivity of 90% to 96%, specificity of 96%, and accuracy of 95%. Newer multidetector CT scanners with multiplanar reformation capability are considered more effective in evaluation of small bowel obstruction. Therefore, CT is considered the best modality for determining which patients would benefit from conservative management and close follow-up and which patients would benefit from immediate surgical intervention.<sup>33</sup> CT criteria for small bowel obstruction are the presence of dilated small bowel loops (diameter >2.5 cm from outer wall to outer wall) proximally to normal-caliber or collapsed loops distally. It should be noted that multidetector CT usually does not require oral contrast material because the retained intraluminal fluid serves as a natural





**FIG. 16.5** (A) Air-fluid levels (*arrows*) consistent with postoperative ileus. (B) Prominent small bowel loops consistent with distal small bowel obstruction (*yellow line*). (C) Computed tomography scan of the abdomen and pelvis. Small bowel obstruction with evidence of transition point (*arrow*).

negative contrast agent, and it allows assessment of extramural areas that would not be visible at contrast-enhanced studies.

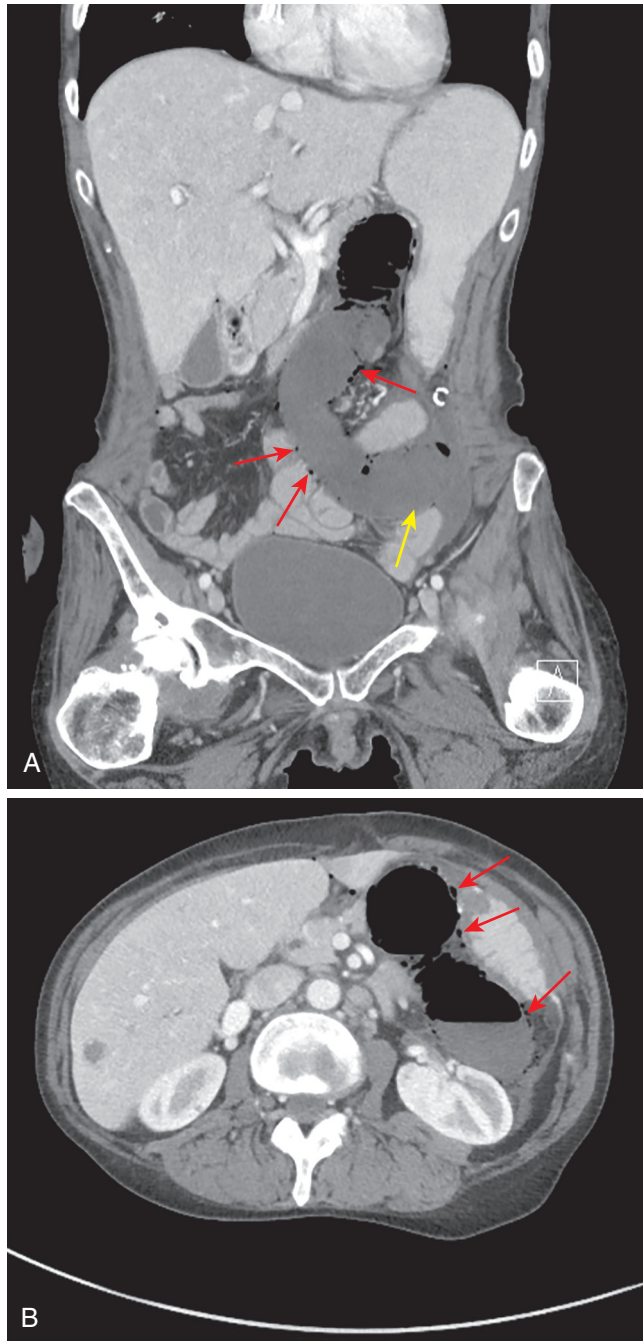
If CT scan is unavailable, sonography can sometimes serve as a useful substitute. Sonography is not commonly used for the evaluation of small bowel obstruction, mainly because most of the time the bowel loops are filled with gas, producing nondiagnostic sonograms, and because adhesions, the most common cause of mechanical small bowel obstruction, are not detected with this technique.<sup>34</sup> However, when the obstructed bowel segments are dilated and filled with fluid, not only can the level of obstruction be recognized but the cause of the obstruction can also be demonstrated with the use of the fluid-filled bowel as a sonic window.

### Treatment

Aggressive intravenous fluid therapy and correction of electrolyte imbalance are crucial in the initial management of acute

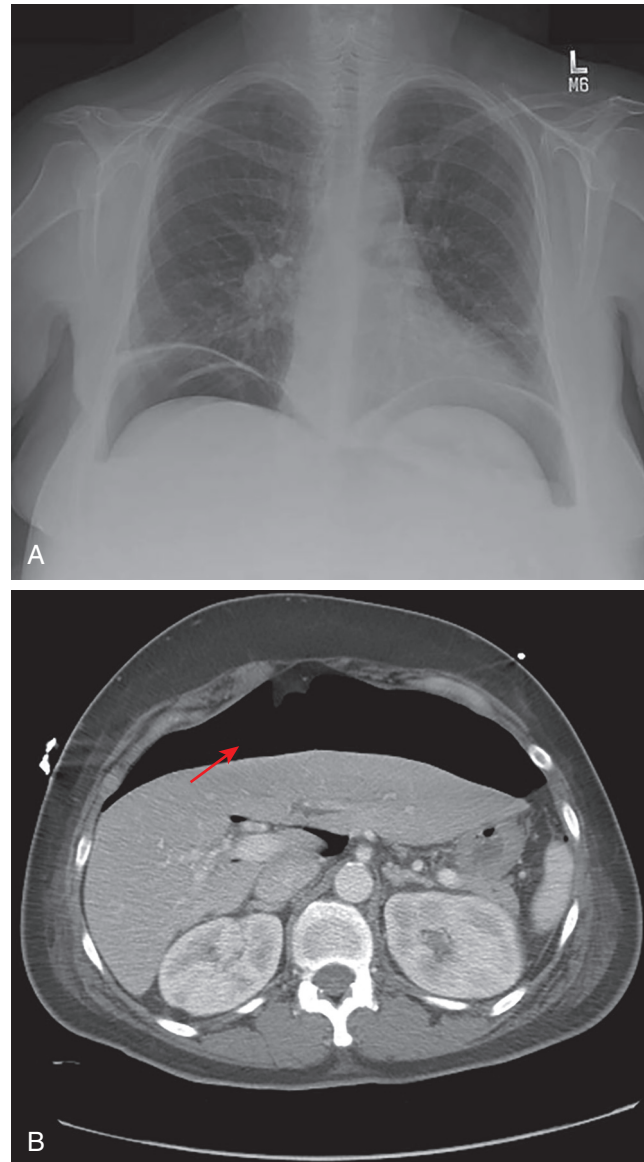
small bowel obstruction. A Foley catheter and occasionally a central venous catheter are needed to monitor fluid resuscitation. Blood tests identify electrolyte imbalance, elevated leukocyte count, abnormal liver function test results, elevated amylase level, acidosis, anemia, and bleeding tendency. A nasogastric tube allows decompression of the stomach and prevents aspiration. Traditionally, it has been recommended that patients with small bowel obstruction (without indications for immediate surgical exploration) should be observed for no longer than 12 to 24 hours, after which time, if no improvement is seen, the patient should undergo exploration. However, as long as there remain no findings on serial clinical evaluation to suggest a complicated obstruction, the patient may be observed for a longer period of time. Repeated examination of the patient during this period is extremely important.

Data regarding nonoperative management suggest it to be successful in 65% to 81% of partial small bowel obstruction



**FIG. 16.6** Computed tomography scan of the abdomen and pelvis (coronal [A] and axial [B] views). Air in small bowel wall consistent with pneumatosis intestinalis (red arrows) and closed-loop obstruction of small bowel (yellow arrow).

cases. All patients suspected of having complicated bowel obstruction (complete obstruction, closed-loop obstruction, bowel ischemia, necrosis, or perforation) based on clinical and radiologic examination should be taken to the operating room for abdominal exploration (Fig. 16.7). It should be noted that for patients who ultimately require an operation, a delay of more than 1 day has been identified as a risk factor for requiring bowel resection<sup>35</sup> (Fig. 16.8).



**FIG. 16.7** Pneumoperitoneum demonstrating free air within abdominal cavity. (A) Abdominal radiograph. (B) Computed tomography scan of the abdomen and pelvis (arrow).

### Anastomotic Leaks

The overall incidence of anastomotic leaks is approximately 2% to 7%.<sup>36</sup> The lowest leak rates are found with ileocolic anastomosis (1%–3%), and the highest rates are found in coloanal anastomosis (10%–20%).<sup>37</sup> The mortality rate for an anastomotic leak in the literature typically is in the 10% to 15% range.<sup>38</sup> In the study by Maggioni and colleagues,<sup>2</sup> the rate of leaks in patients undergoing pelvic exenteration was 2.8%. In the setting of pelvic exenteration, the anastomotic leak usually occurs as a result of small bowel anastomosis when a segment of ileum is used as the incontinent urostomy. The anastomotic leak may also be seen in the setting of ileocolonic anastomosis when a continent conduit is performed after the distal ileum and ascending colon have been used for the urinary conduit.

Most anastomotic leaks usually become apparent 5 to 7 days postoperatively. The majority of the literature defines