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Unexpected Gynecological Findings during Abdominal Surgery

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INTRODUCTION

As general surgeons, we are often asked to evaluate acute abdominal pain. The differential diagnosis of abdominal pain is broad, and includes disorders of the gastrointestinal, urogenital, gynecologic, vascular, and pulmonary systems. Abdominal pain may be caused by infectious, inflammatory, anatomic, or neoplastic processes. The management of abdominal pain varies by etiology, and accurate diagnosis is key to avoiding inappropriate treatment.

Specifically, abdominal pain in women presents an additional diagnostic dilemma. Disease processes found exclusively in women should be kept in mind when presented with a female patient with abdominal pain. In women of childbearing age, pregnancy and conditions causing acute abdominal pain only during pregnancy must be considered, and all women of childbearing age should have either a serum or urine β -human chorionic gonadotropin (HCG) level as part of the initial diagnostic workup. In a pregnant patient, the differential diagnosis is modified, and should include disorders specific to pregnancy.

Gynecological disorders that specifically occur during pregnancy include ectopic pregnancy, threatened abortion, retroverted gravid uterus, spontaneous uterine rupture, and chorioamnionitis. In addition, there are disease processes that are not specific to, but frequently occur during pregnancy. These may include torsion of a normal ovary or ovarian cyst or mass, pyelonephritis, pelvic inflammatory disease, acute salpingitis, tubo-ovarian abscess, pyosalpinx, torsion or degeneration of a uterine fibroid, and hemorrhage or rupture of an ovarian cyst. Accurate recognition and treatment of the disease is integral in order to achieve the best outcome for both the mother and developing fetus.

Finally, both pregnant and non-pregnant women experience typical general surgical problems, including acute cholecystitis, appendicitis, diverticulitis, bowel obstruction, and perforated ulcers. However, the treatment of these diseases may be modified during pregnancy. For example, acute cholecystitis is often managed non-operatively during the first and third trimesters of pregnancy, while surgical intervention is recommended in the second trimester. For other conditions, surgical intervention is recommended regardless of gestational age. For instance, given the risk of rupture with acute appendicitis during pregnancy, appendectomy is indicated regardless of gestational age.

Acute appendicitis is the most common surgical emergency,¹ and is also the most common cause of non-gynecological pelvic pain.² Many gynecologic conditions can mimic acute

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appendicitis, making the diagnosis unclear. Pelvic pathology may also be confused with other intraabdominal disease processes. For example, diverticulitis may be mimicked by an ovarian cyst or tubo-ovarian abscess; pelvic inflammatory disease may be misdiagnosed as generalized peritonitis secondary to a perforated viscera; acute cholecystitis may be confused with ovarian, appendiceal, or uterine pathology in the right upper quadrant during pregnancy. Despite vast improvements in imaging over the last three decades, at times it may still be difficult to differentiate between gynecologic and nongynecologic causes of abdominal pain prior to laparotomy or laparoscopy.

The first step in the evaluation of a woman with abdominal or pelvic pain should include a complete history and physical examination. History-taking should include the history of present illness and characterization of the abdominal pain, medical and surgical history (in particular, previous pelvic surgeries, including hysterectomy and oophorectomy), sexual and contraceptive histories, and last menstrual period. Physical exam should include abdominal, pelvic, and bimanual examinations. In particular, careful questioning about and inspection for vaginal discharge or bleeding on pelvic exam should be performed. Physicians are commonly taught that cervical motion tenderness is pathognomonic for pelvic inflammatory disease (PID). However, any disease process causing pelvic inflammation may result in cervical motion tenderness, and other diagnoses should not be excluded based on the presence of this sign.³

In women of childbearing age who have not had a hysterectomy, including those on contraceptives, those with an intrauterine device in place, and those with partners who have had a vasectomy, a pregnancy test via urine β -HCG should be performed. When the diagnosis is unclear and a gynecological cause is included in the differential diagnosis, a gynecology consult should be considered. In all pregnant patients with abdominal pain, a gynecology consult is strongly recommended to optimize fetal and maternal outcomes. If diagnostic uncertainty exists, laparoscopy with direct visualization of the pelvis and abdominal cavity is often the best modality for investigating pelvic pain in women.

The goal of this chapter is to discuss common obstetric and gynecologic abnormalities which the general surgeon may encounter in the evaluation of the female patient with acute abdominal pain. We will differentiate those gynecologic conditions that are specific to pregnancy and those that are less common during pregnancy. As many of these conditions can be diagnosed preoperatively, the diagnostic workup for each will be discussed. In cases where the diagnosis is unclear or recognized intraoperatively, we will discuss the surgical management of each entity. At times, pelvic pathology is incidentally found during elective abdominal procedures for other diagnoses; in this case, the appropriate intraoperative management of incidentally recognized obstetrical/gynecological pathology will be discussed.

ACUTE ABDOMINAL PAIN IN THE FEMALE PATIENT

Consider a 24-year-old female presenting with a 48 hour history of abdominal pain localizing to the right lower quadrant. She reports fever to 102°F with associated nausea and vomiting. On physical exam, she is tender in the bilateral lower quadrants, right greater than left. While the story is consistent with acute appendicitis, the differential diagnosis is broad and includes PID tubo-ovarian abscess, pyosalpinx, ectopic pregnancy, endometriosis, ruptured or hemorrhagic ovarian cyst, ovarian torsion (of normal ovary, ovarian cyst, or ovarian mass), and torsion or degeneration of a uterine fibroid. A comprehensive history and physical are the first steps in differentiating the cause of the abdominal pain. This will guide your differential diagnosis. The age of the patient, menstrual history, and sexual history will

further refine your differential diagnosis. Table 1 compares the signs and symptoms of acute appendicitis with those of common gynecological diseases causing acute abdominal pain.

Acute appendicitis accounts for 27.5% of abdominal surgical emergencies, making it the most common surgical emergency.¹ Acute appendicitis is also the most common cause of non-gynecological pelvic pain.² Appendicitis can occur at any age, although the mean age at presentation is 53.5 years.⁴ Approximately 10% of patients with acute appendicitis are less than 10 years of age, while an additional 10% are older than 50 years of age.⁵ Perforation of the appendix occurs in approximately 14-21% of adult patients with acute appendicitis; children and the elderly have higher rates of perforated appendicitis.⁵⁻⁷

Many of the signs and symptoms of acute appendicitis overlap with those of the gynecologic disorders on the differential. In fact, acute appendicitis has been shown to occur simultaneously with a variety of gynecologic diseases, including endometriosis, ovarian cysts, uterine fibroids, and hydrosalpinx.⁸ Because of this, the misdiagnosis rate of appendicitis in women of reproductive age has increased over time.⁹ The most common symptom of acute appendicitis is abdominal pain. A history of mid-epigastric pain migrating to the right lower quadrant is classic, and has a positive predictive value of 90% in adults with acute appendicitis. Nausea, vomiting, diarrhea, and anorexia are also common.^{3, 5, 6} Fever and leukocytosis may be present, and have been reported in approximately one-third of adults with acute appendicitis. However, many of these signs and symptoms may not be helpful in distinguishing acute appendicitis from PID.⁵

On physical exam, patients with acute appendicitis have abdominal tenderness, localized to the right lower quadrant in over 90% of cases. Other signs of peritoneal irritation, including guarding and rebound tenderness, may be elicited.⁵ Rovsing's sign (pain in the right lower quadrant with palpation of the left lower quadrant), the psoas sign (pain with right hip extension), and the obturator sign (pain with flexion and internal rotation of the right hip) may also be elicited on physical exam. On speculum exam, cervical motion tenderness may be present, particularly if the appendix is located in the pelvis. Purulent fluid in the pelvis or a pelvic abscess from perforated appendicitis may also cause cervical motion tenderness. However, each of these findings can be found with PID or a tubo-ovarian abscess. Vaginal discharge should not be present in the case of acute appendicitis, but an undiagnosed sexually transmitted disease can coexist with appendicitis or any other abdominal pathology.

Computed tomography (CT) is the imaging modality of choice for suspected acute appendicitis in adults.² CT findings consistent with appendicitis include periappendiceal fat stranding, an enlarged, appendix that does not fill with contrast, and cecal/appendiceal wall thickening.^{1, 4, 10} In a retrospective review of patients with acute appendicitis and tubo-ovarian abscess, patients with acute appendicitis were more likely to have an enlarged appendix (≥ 6 mm), a thickened appendiceal wall (≥ 3 mm), periappendiceal fluid collection, cecal wall thickening, and pericecal fat stranding on CT scan.⁴ Interestingly, female patients who began their menstrual period within seven days of presentation with abdominal pain were five times more likely to have pelvic inflammatory disease than acute appendicitis.⁵

Transvaginal and transabdominal ultrasound may also be obtained when the diagnosis is unclear or CT scanning is contraindicated. Ultrasound findings consistent with appendicitis include a non-compressible, non-peristaltic tubular structure arising from the base of the cecum measuring more than 6 mm in diameter.^{2, 11} An appendicolith may also cause acoustic shadowing on ultrasound.¹¹ The transvaginal component tends to be more sensitive and specific than transabdominal ultrasound when evaluating for gynecological pathology.¹²⁻¹⁵ In addition, ultrasound is essential in the woman with a positive urine β -HCG to document intrauterine pregnancy and approximate gestational age.

Appendectomy is the standard of care for uncomplicated appendicitis either via open or laparoscopic route. This is true regardless of pregnancy status and gestational age, though the maternal and fetal risks differ with the latter. Gestational age may affect the choice of approach, with laparoscopy not recommended after 20 weeks gestation. Reported negative appendectomy rates can be as high as 25%, in adult women, higher than that in both men and children.^{5, 6} Furthermore, misdiagnosis of appendicitis has increased over time in women of reproductive age.⁹ Laparoscopic intervention allows for direct visualization of the abdominal cavity if the diagnosis is unclear, and has been shown to dramatically reduce the negative appendectomy rate, particularly in women of reproductive age, in which other etiologies are common.^{16, 17}

When acute appendicitis is suspected but the appendix appears normal at surgery, the management of the appendix at laparotomy or laparoscopy is controversial. For patients undergoing open appendectomy through a McBurney or Rocky-Davis incision, appendectomy is recommended to prevent diagnostic confusion in the future. However, appendectomy is more controversial in the case of laparoscopy. In cases where the cause of abdominal pain can be identified during laparoscopy (adnexal torsion, ovarian mass, tubo-ovarian abscess, PID), some surgeons feel that the appendix should be left in place. Others report high rates of pathologically-confirmed appendicitis when appendectomy was performed incidentally during laparoscopic surgery for gynecologic disease and recommend incidental appendectomy in female patients undergoing laparoscopy to prevent diagnostic confusion or future episodes of appendicitis.⁸ Appendectomy should be considered when no other definitive cause of abdominal pain is recognized.

Acute appendicitis occurring during pregnancy deserves special mention. Acute appendicitis is the most common reason for surgical intervention during pregnancy.¹⁸ However, diagnosis during pregnancy is commonly delayed. The appendix may be displaced upward during pregnancy due to a gravid uterus, making the typical location of right lower quadrant pain unreliable for diagnosis. Gastrointestinal complaints such as abdominal pain and nausea may be attributed to pregnancy rather than appendicitis. In addition, physiological leukocytosis is common during pregnancy.^{2, 18} The rate of appendiceal perforation has been reported to be as high as 40% during pregnancy due to delayed diagnosis.¹⁸ With rupture of the appendix, the rate of fetal loss may be as high as 24%, compared to 7-10% in unruptured appendicitis during pregnancy.¹⁸

Ultrasound is the imaging modality of choice for suspected appendicitis in pregnancy, and provides the best visualization during the first and second trimester. Ultrasound may be less sensitive during the third trimester, as the enlarging uterus causes migration and rotation of the appendix.¹¹ Magnetic resonance imaging (MRI) may be used during pregnancy when ultrasound results are inconclusive,¹¹ and has been shown to have a sensitivity of 80% and specificity of 99% during pregnancy.¹⁸ MRI findings with appendicitis are similar to those seen on CT and include appendiceal wall thickening (>3 mm), increased appendiceal diameter (≥6 mm), a fluid-filled appendiceal lumen, and periappendiceal fat stranding.^{2, 11} Gadolinium contrast should be avoided during pregnancy.^{11, 19} Currently, the American College of Obstetricians and Gynecologists recommends the second trimester as the optimal time for non-obstetric surgery. However, in the case of emergent surgery or maternal risk, surgical intervention should be undertaken, regardless of gestational age.²⁰

Although acute appendicitis is the most frequently encountered non-gynecologic cause of abdominal pain, there are several gynecologic pathologies that may also be encountered in a non-pregnant patient. These include pelvic inflammatory disease with or without tubo-ovarian abscess, endometriosis, ruptured or hemorrhagic ovarian cyst, adnexal torsion, uterine fibroids, and bicornuate uterus.

GYNECOLOGICAL CAUSES OF PELVIC/ABDOMINAL PAIN IN THE NON-PREGNANT PATIENT

Pelvic Inflammatory Disease (PID)

PID is defined as the infection and inflammation of the upper female genital tract, including the cervix (cervicitis), fallopian tubes (salpingitis), and uterine lining (endometritis).²¹ Peritonitis may also be present.^{22, 23} PID is thought to be caused by an ascending infection from the lower genital tract, and as such, is a common complication of sexually transmitted diseases in women.²³ Less importance has been placed on determining the exact location of the inflammation, but rather the early diagnosis and treatment to prevent long-term morbidity in these patients.²² An episode of acute PID can result in recurrent episodes of PID, chronic pelvic pain, ectopic pregnancy, and infertility.²³⁻²⁵

PID frequently occurs in women of reproductive age, with the majority of cases occurring in women aged 20-40.^{23, 24} The mean age at diagnosis is 27 years. One in nine women will be diagnosed with PID during their reproductive years.²⁶ Risk factors include a history of prior episodes of PID, multiple sexual partners, black race, intrauterine device (IUD) use, substance abuse, low socioeconomic status, and lack of contraceptive use.^{24, 26-28}

Microorganisms commonly associated with PID include *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, diphtheroids, *Gardnerella vaginalis*, *Mycoplasma genitalium*, *Prevotella*, *Bacteroides*, anaerobes, and streptococci.^{21, 23, 24, 29} Although originally thought to be attributed to *N. gonorrhoeae* and *C. trachomatis* primarily, it is now recognized that only two-thirds of cases of PID are caused by these organisms. Approximately 30% of cases can be attributed to anaerobes or aerobes only without evidence of *N. gonorrhoeae* or *C. trachomatis*.²³ More than half of PID cases have more than one microorganism isolated.²¹

Symptoms of PID can overlap with acute appendicitis and include abdominal pain, nausea and vomiting, vaginal discharge, abnormal vaginal bleeding, urinary frequency, and back pain.^{21, 22, 24} Fever may be present,²⁴ but can be unreliable, as more than 50% of patients admitted with PID do not meet the clinical definition of fever.²⁶ Similarly, elevated white blood cell count (WBC), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and CA-125 are non-specific findings in PID.^{22, 26} On physical examination, patients with PID typically demonstrate abdominal and pelvic tenderness. On bimanual exam, adnexal and cervical motion tenderness may be elicited, and, in contrast to acute appendicitis, mucoopurulent cervicovaginal discharge may be visualized.^{2, 21, 24, 30} As the signs and symptoms of PID can be vague and misleading, the diagnosis of PID can be missed in up to 35% of patients.²

Because of this, radiologic imaging may be employed when the diagnosis is unclear or the patient is not responding to treatment, with ultrasound being the modality of choice. In early or uncomplicated PID, sonographic findings may be unremarkable. Findings of PID on ultrasound are typically bilateral but can be asymmetric. Transvaginal ultrasound is more sensitive than transabdominal ultrasound in detecting abnormalities in the uterus and adnexa. The uterus may be enlarged with a thickened and indistinct endometrium. Debris may be visualized in the endometrial cavity or the pelvis. The ovaries may also be enlarged due to reactive inflammation.^{2, 30, 31} When the fallopian tubes are involved, they may appear edematous, fluid-filled, and distended on ultrasound.^{2, 22, 30, 31} Chronic inflammation of the fallopian tubes may lead to a "beads on a string appearance."³⁰ Hypervascularity and hyperemia of the fallopian tubes can be seen on Doppler ultrasound.^{2, 30}

While ultrasound is the diagnostic modality of choice for PID, CT scanning can be used, especially during the acute presentation when the diagnosis may be unclear and other

intraabdominal pathology may be suspected.²² Findings consistent with PID on CT scan include pelvic inflammation and fat stranding, endometrial fluid and debris, and indistinct tissue planes.^{2, 22, 30, 31} With advanced PID, bowel obstruction, hydronephrosis or hydroureter, and right upper quadrant inflammation may be visualized.²²

Consider the 24 year old patient presented earlier. CT scan was done for the suspicion of acute appendicitis. On physical exam, she had cervical motion tenderness. She was sexually active and not pregnant. CT scan demonstrated an enlarged, fluid-filled tubular structure in the pelvis, remote from the cecum (Figure 1A), with more distal cuts showing a right adnexal fluid collection and a left ovarian cyst (Figure 1B). Given the diagnostic uncertainty, pelvic ultrasound was also performed, demonstrating a dilated, peristaltic tubular structure posterior to the right ovary, a left ovarian cyst, and free fluid in the pelvis. Even after gynecological consultation, the diagnosis was unclear with appendicitis, PID, and tubo-ovarian abscess included in the differential diagnosis. The decision was made to proceed with laparoscopy and she was found to have perforated appendicitis with the appendix and abscess located down in the pelvis near her right adnexa.

MRI can be useful in diagnosing PID, and may be more accurate than ultrasound in certain patients.^{2, 22} In particular, MRI has a high sensitivity for diagnosing pyosalpinx, which is seen as a hyperintense tubular structure on T2-weighted images. Pelvic fat stranding and enhancement of the fallopian tube walls may also be present. However, MRI is not used as a first line modality in diagnosing PID, as ultrasound and CT are more cost-effective and readily available.^{2, 31}

As the signs, symptoms, and imaging findings of PID can be indistinct, surgical intervention may be undertaken when the diagnosis is not yet clear. Laparoscopy can be used to directly visualize pelvic anatomy and diagnose PID without aggravating the inflammatory process. Specifically, the criteria for diagnosing salpingitis on laparoscopy include edema of the wall of the fallopian tube, hyperemia of the fallopian tube surface, and an exudate from the ends or surface of the fallopian tube. However, in patients with endometritis without salpingitis, the diagnosis may be missed. Currently, due to its invasive nature, laparoscopy is not routinely used for the diagnosis of PID, and should be reserved for cases in which the diagnosis is unclear or complications of PID are suspected.^{22, 31}

Treatment of PID consists primarily of antibiotic therapy. Currently, the Centers for Disease Control (CDC) recommend empiric, broad-spectrum coverage that is effective against *N. gonorrhoeae*, *C. trachomatis*, and anaerobes, regardless of their presence on endocervical culture.²³ The recommended treatment regimen for parenteral therapy for PID includes cefotetan or cefoxitin plus doxycycline, or clindamycin plus gentamicin. Levofloxacin with metronidazole and doxycycline may also be used. Oral therapy should include levofloxacin plus metronidazole or a cephalosporin with doxycycline and metronidazole. Success rates with both regimens range from 75-100%.²³ Antibiotics should be continued for 14 days, regardless of route.²⁹ Long-term outcomes of PID recurrence, chronic pelvic pain, ectopic pregnancy, fallopian tube obstruction, and pregnancy are similar with inpatient and outpatient treatments. Outpatient management may be preferred due to potential cost savings.²⁴ Once diagnosed, the gynecologists should make this decision and assume management of the patient.

If PID is diagnosed at the time of laparoscopy for suspected appendicitis or pelvic pain of unclear etiology, the management depends on the severity of the findings and certainty of the diagnosis. PID without tubo-ovarian abscess should be treated with pelvic irrigation and antibiotic therapy as previously discussed. In mild cases of PID without abscess,

appendectomy should be considered to prevent diagnostic confusion in the future. However, in the setting of severe, purulent PID, this is likely not the best course of action.

Fitz-Hugh-Curtis syndrome occurs when the pelvic inflammation of PID spreads to the right upper quadrant via the right paracolic gutter. The infection then involves the peritoneal surfaces, including the liver, resulting in perihepatitis. In the acute setting, patients with Fitz-Hugh-Curtis syndrome present with a history of PID and right upper quadrant pain, which may be confused with acute cholecystitis or hepatitis. Fever and leukocytosis may or may not be present, and elevated CRP and ESR are common. In this case, the diagnosis may not be clear on ultrasound. On CT scanning, perihepatic inflammation and liver capsular enhancement can be seen. Gallbladder wall thickening and perihepatic and pericholecystic fluid can also be present. MRI may demonstrate hypervascularity in the right upper quadrant. A “violin string” adhesion stretching from the peritoneum to the anterior surface of the liver may be seen at the time of surgery (Figure 2). In the non-acute setting, Fitz-Hugh-Curtis syndrome is commonly encountered incidentally during laparoscopy for alternative diagnoses, most commonly laparoscopic cholecystectomy.^{22, 31, 32} When present, care should be taken in retracting the gallbladder to expose the triangle of Calot, as the adhesions of the liver to the peritoneal surface can cause liver lacerations if not freed before attempting retraction.

Tubo-ovarian Abscess (Figure 3)

Tubo-ovarian abscess (TOA) is a serious complication of pelvic inflammatory disease (PID), occurring in approximately 5-34% of women with PID.^{28, 33-35} Similar to PID, TOA typically occurs in women of reproductive age, most frequently in women aged 20-40.^{28, 33, 36} A small percentage of TOAs occur in postmenopausal women.^{27, 36} As many as 60% of women with TOA are nulliparous.²⁸ Risk factors for TOA are similar to those for PID, including multiple sexual partners, intrauterine device (IUD) use, low socioeconomic status, lack of contraceptive use, immunosuppression, and HIV.^{27, 28, 33-35} Approximately two-thirds of TOAs are unilateral, which may lead to misdiagnosis as acute appendicitis when right-sided.^{28, 33-36}

The etiology of TOA is related to an initial insult to the female genital tract. In particular, inoculation of the fallopian tube epithelium with a pathogen has been proposed as the initial step in formation of TOA.^{27, 34, 35} Destruction of the fallopian tube produces a purulent exudate, and the resulting low oxygen environment is favorable for anaerobic organisms.^{27, 34, 35} The subsequent inflammatory response induces edema, ischemia, and necrosis of the fallopian tube. Surrounding structures such as the ovary, round ligament, broad ligament, contralateral fallopian tube and ovary, appendix, bowel, and bladder may become involved in the expanding inflammation, and a walled-off abscess develops.^{27, 34} With an expanding infection, rupture of the TOA may occur.^{27, 28, 34} Interestingly, TOA may be the result of non-gynecologic diseases, including diverticulitis, appendicitis, inflammatory bowel disease, and surgery.³⁶

The infectious etiology of TOA is typically polymicrobial. The most commonly identified organisms associated with TOA are *Escherichia coli*, *Bacteroides fragilis* and other *Bacteroides* species, peptostreptococci, peptococci, *Enterococcus*, *Klebsiella*, *Staphylococcus*, *Streptococcus*, and *Hemophilus influenza*.^{35, 36} Although *Neisseria gonorrhoeae* and *Chlamydia trachomatis* are well known causes of PID, these organisms are rarely cultured from TOAs.^{27, 28, 35, 36} Anaerobic bacteria are present in 60-100% of TOA cultures.^{27, 35}

The symptoms of TOA are similar to those of PID and appendicitis and include lower abdominal pain (acute or chronic), nausea, and vomiting.^{27, 28, 34-36} Fever and chills may be

present, but are unreliable, as up to 50% of patients with TOA can be afebrile.^{33, 35} Patients may also report a history of vaginal discharge or abnormal vaginal bleeding.³⁵ A history of PID may be present.^{27, 33-35} When the bowel is involved, patients may report a history of anorexia or diarrhea.²⁷ Leukocytosis may be present but is not a reliable indicator of TOA.³⁵

On physical examination, an abdominal or pelvic mass may be palpated.³⁶ Rebound tenderness or guarding may be elicited on abdominal exam.^{27, 28} Cervical motion tenderness on speculum exam is common.^{28, 34, 36} Mucopurulent cervicovaginal discharge should point the physician away from a diagnosis of appendicitis and toward a diagnosis of PID or TOA.^{27, 28, 34}

Ultrasound is the diagnostic imaging modality of choice for TOA. Ultrasound has a sensitivity of greater than 90% in diagnosing TOA.^{13, 35} Transabdominal ultrasound provides a larger field of view and may be helpful in identifying adnexal masses, although transvaginal ultrasound may provide a more detailed view of pelvic anatomy and vasculature.^{13, 34} On ultrasound, a TOA appears as a complex, multilocular, cystic mass with thickened, irregular walls. Internal echoes and debris and fluid-fluid levels may be present within the collection.^{1, 2, 27, 28, 30, 34, 35} Tubal and ovarian architecture is typically distorted, with destruction of the planes between the ovary and the developing abscess.^{2, 30} Hyperechoic purulent fluid may be visualized in the pelvis.³⁰ A “cogwheel sign” may be seen due to thickening of the endosalpingeal folds.^{2, 34}

CT scanning may be indicated when the diagnosis is unclear. CT scan findings indicative of TOA include a septated, tubular structure with thickened walls.^{2, 27, 28} A dilated, fluid-filled fallopian tube may appear as an adjacent “serpiginous” structure, which may be difficult to differentiate from an inflamed pelvic appendix (Figure 1).² When surrounding tissues are involved in the inflammation, hydronephrosis or hydroureter may be seen.²⁷ The presence of gas bubbles within the fluid collection is highly specific for TOA.^{2, 27} In a large study comparing the CT findings of TOA and acute appendicitis, TOA was highly associated with the appearance of an abnormal ovary, peri-ovarian fat stranding, small bowel and recto-sigmoid wall thickening, and free fluid in the pelvis.⁴

MRI imaging is used infrequently in the diagnosis of TOA. MRI may demonstrate a round adnexal mass with thickened walls and low signal density on T1-weighted images. Surrounding tissue inflammation can be seen.^{2, 34} MRI has been reported to have a sensitivity and specificity of 100% and 90%, respectively, in diagnosing TOA.¹³ Ultrasound and CT scanning are more readily available and cost effective and MRI should be reserved for cases where the diagnosis is uncertain.²

The goal of treatment for TOA is to treat the associated infection and preserve fertility, whenever possible. The mainstay of treatment for TOA is antibiotic therapy, with or without additional surgical or drainage procedures. Antibiotic therapy should be specific to the organisms most common in TOA, including anaerobes. Current recommendations are for the use of clindamycin plus an aminoglycoside or cefoxitin plus doxycycline.^{27, 28, 37} Antibiotics should be continued parenterally until the patient is afebrile for 48 hours, with continuation of oral antibiotics for 14 days.^{28, 37} Options for oral antibiotics include levofloxacin or doxycycline plus metronidazole.³⁷ Response rates with medical management alone range from 42-100%. Patients who receive primary antibiotic therapy have longer hospital stays, longer duration of fever, and require surgical intervention more frequently compared to patients who undergo a drainage procedure initially.^{28, 33}

Currently, surgical intervention is recommended for three situations in patients with TOA: 1) when there is concern for an alternative surgical emergency, such as appendicitis, cholecystitis, bowel obstruction, or bowel perforation, 2) failure of clinical response after

48-72 hours of medical management, and 3) intraabdominal rupture of TOA, resulting in peritonitis and a surgical emergency.²⁷ For the last two, a diagnosis has already been determined and a gynecologist is typically managing the patients and making decisions regarding surgical intervention. As general surgeons, we are most likely to encounter a TOA when operating for suspected appendicitis or other intraabdominal pathology. Given that patients who undergo initial drainage have shorter hospital stays and less morbidity, drainage of the TOA diagnosed at laparoscopy or laparotomy is indicated.

Historically, aggressive surgical therapy was the primary treatment modality for TOA, with procedures ranging from adnexectomy to total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO). However, due to high rates of bowel injury³⁴ and concern for future fertility and maintenance of hormone function, conservative surgical management is advised, with the mantra “less is more” being employed. A complete inspection of the abdominal cavity is necessary, including the uterus, bilateral ovaries and fallopian tubes, appendix, sigmoid colon, and upper abdomen. Lysis of adhesions should be performed, taking care to protect the nearby ureter. Samples of the abscess fluid should be sent for culture and sensitivity. The pelvis should be irrigated copiously, and the placement of closed-suction drains should be considered. In the case of frank contamination of the abdominal cavity, the skin and subcutaneous tissues should be left open to close by secondary intention.²⁷

Patients who have undergone at least 48 hours of medical management with appropriate antibiotics and have failed to achieve a clinical response are candidates for surgical intervention. Typically, a positive clinical response is defined as relief of pain, normalization of body temperature, decrease in leukocytosis, and improvement in sonographic features of the TOA.³⁶ This will most likely be done by gynecological surgeons, but in some cases a general surgeon may need to do this. When surgery is indicated, laparotomy or laparoscopy can be performed. Laparotomy may be difficult due to the presence of inflammation and adhesions in the abdominal cavity.³³ The advent of laparoscopic surgery has allowed for a minimally invasive surgical approach to TOA. Laparoscopic lysis of adhesions and drainage of the TOA can be performed safely, with a complete response rate of greater than 90%.^{27, 28, 34, 38} When possible, laparoscopy is preferred over laparotomy in patients with TOA who require surgical intervention.

Other options for failure of medical management include percutaneous drainage of the TOA cavity via an image-guided transabdominal or transvaginal approach. Success rates range from 83-100% when percutaneous transvaginal drainage is performed concomitantly with antibiotic administration.³⁴ In addition, drainage can decrease the length of hospital stay and reduce the duration of fever in patients with TOA. Percutaneous drainage can also be utilized as salvage therapy for patients who have failed medical management, with a response rate of 95%. Patients who fail medical therapy are typically older and have a larger total TOA volume when compared to patients who respond to antibiotics alone.³³

The transabdominal route is preferred for drainage of abdominal and upper pelvic abscesses and can be performed under ultrasound or CT guidance.³⁴ CT guidance can also be used for a transgluteal approach.²⁷ Transvaginal drainage of TOA is preferred for low-lying pelvic abscesses.³⁴ The transvaginal approach allows for direct access to the adnexal area from the vagina. Gjelland et al. described a large series of women who underwent ultrasound-guided transvaginal drainage of TOA. Two-thirds of the patients required one drainage procedure, with an additional 27% achieving a complete response after a second drainage procedure. Half of the patients did not require anesthesia or analgesia, indicating that this procedure causes minimal discomfort to patients. Transvaginal drainage was successful in 93.4% of patients, and there were no complications.³⁶ Similarly, Gohorkhay et al. reported a complete

response rate in 100% of patients who underwent percutaneous drainage initially for TOA.³³ An indwelling catheter may be left in place after a percutaneous drainage procedure.

A known complication of TOA is intraabdominal rupture, which occurs in 3-15% of cases.^{28, 35} In the case of a ruptured TOA, surgical intervention should be undertaken immediately. At times, patients may be taken urgently to the operating room due to hemodynamic instability but without a definitive diagnosis. Sepsis and multiple organ failure develop rapidly in these patients if left untreated.²⁷ If encountered by the general surgeon at laparotomy, gynecology consultation is recommended, but in the absence of gynecology availability, unilateral salpingo-oophorectomy is indicated.

Approximately 1-6% of cases of TOA occur in postmenopausal women.^{27, 36} When a postmenopausal patient presents with TOA, malignancy should be suspected and a complete workup initiated. It is strongly recommended that an exploratory laparotomy be performed in these patients to exclude the diagnosis of concurrent intraabdominal malignancy.^{27, 34}

Endometriosis

Endometriosis is a common cause of abdominal and pelvic pain in women. In addition, it can mimic acute general surgical disease processes such as appendicitis and cause general surgical problems such as small or large bowel obstruction. Endometriosis is defined as the presence of endometrial glands and stroma outside of the uterine cavity.³⁹⁻⁴³ Endometriosis is most common in women of reproductive age, with one large study reporting a mean age at diagnosis of 35 years.⁴¹ Several large reviews have estimated the prevalence of endometriosis to be 7-10% in the general population and up to 50% in infertile women. Women struggling with infertility are up to eight times more likely to have endometriosis than fertile women.^{42, 43} Additionally, endometriosis is estimated to occur in up to 60% of women with dysmenorrhea and up to 87% of women with chronic pelvic pain.^{42, 44} The incidence of endometriosis may also be increasing over time.⁴¹ Symptoms of endometriosis include dysmenorrhea, dyspareunia, chronic pelvic pain, and pain associated with ovulation, micturition, and defecation. Pain may be cyclical and related to the menstrual cycle.⁴⁴ In fact, appendicitis has been shown to occur simultaneously with endometriosis in women undergoing laparoscopy, which may complicate the diagnosis.⁸

Risk factors for endometriosis are multiple and varied. Ozkan et al. reviewed the evidence, and suggest that there may be a genetic component to the development of endometriosis, as identical twins have been shown to have the disease. In addition, Asian women may be at higher risk, while black women are at lower risk. An early onset of menarche (≤ 11 years of age), short menstrual cycles (≤ 27 days), reduced parity, and heavy or prolonged menstrual bleeding have been associated with an increased risk of endometriosis. Tall, thin women have been shown to have endometriosis more frequently, while increased body weight and BMI may decrease the risk of endometriosis. Exercising more than four hours per week may decrease the risk of endometriosis, while excess caffeine or alcohol intake has been associated with increased rates. Interestingly, smoking has been associated with decreased risk of endometriosis.⁴³

The American Society for Reproductive Medicine has classified endometriosis into four stages. Points are given based on the size (<1 , 1-3, >3 cm) and depth of invasion (superficial, deep) of endometriotic lesions, and the extent and consistency (filmy, dense) of abdominal adhesions. Stage I is defined as minimal (1-5 points), Stage II as mild (6-15 points), Stage III as moderate (16-40 points), and Stage IV as severe (>40 points).⁴⁵ In a large study of women with endometriosis, 37% were diagnosed with Stage I or II, while 63% were diagnosed with Stage III or IV endometriosis. The most common location for endometrial implants overall is the ovaries, followed by the deep and central pelvis,

appendages, and vesicouterine pouch.⁴¹ Almost 60% of cases of Stage IV endometriosis involve the intestinal tract (rectum, sigmoid, colon, appendix, small bowel), while 13.8% of cases involve the urinary tract (bladder, ureters).^{30, 46}

The most accepted theory of the development of endometriosis is that of retrograde menstruation. Other theories include coelemic metaplasia, lymphatic or vascular embolization of endometrial tissue, and transformation of embryonic rests.^{39, 40, 42} Endometrial implants tend to occur asymmetrically in the abdomen, being located on the left more frequently than the right.⁴⁶ The presence of endometrial tissue causes inflammation, which results in pain, adhesions, and pelvic anatomic distortion. In women with Stage IV disease, pain may be mediated by the presence of deep infiltrating endometrial lesions in the muscularis propria of surrounding organs.^{40, 47}

General surgeons will encounter endometriosis in one of three situations: 1) as an incidental finding during laparotomy or laparoscopy for another reason, 2) as the cause of acute abdominal pathology such as small bowel obstruction (Figure 4), or 3) when consulted by gynecology intraoperatively or preoperatively for stage IV disease involving the bowel or other intraabdominal structures. When found incidentally during abdominal surgery in an asymptomatic patient, treatment for endometriosis should be deferred and the patient should be referred to a gynecologist for definitive management. However, if the physician feels that the implant is causing adhesions or may cause potential problems, resection is indicated if it is safe in the context of the planned operations. For example, a solitary endometrioma on the small bowel that is partially obstructing might be resected in an elective case for another reason, but not in an emergent operation for peritonitis from another etiology. An additional indication for resection is diagnostic uncertainty, especially in the setting of an operation done for an abdominal malignancy, in which a malignant implant needs to be definitively ruled out.

In the second case, endometriosis may cause an acute problem such as small bowel obstruction (Figure 4). In these cases, the treatment of the bowel obstruction, usually with resection of the involved segment, is indicated. In emergent cases where an adequate bowel prep is not feasible, ileostomy or colostomy may be necessary. Multidisciplinary consultation may be needed for multi-visceral involvement.

When consulted to assist gynecology in managing complex endometriosis, the surgical management is variable, but several studies have demonstrated the effectiveness of surgery in the relief of endometriosis-related pain. The goal of surgical intervention for endometriosis is the removal or ablation of visible endometrial implants and the restoration of normal pelvic anatomy. Laparoscopy is currently recommended, as patients have a faster return to work, decreased duration of hospital stay, and less pain.⁴³ However, laparotomy may be required for severe cases where significant bowel or bladder involvement is present. In these cases, preoperative multidisciplinary consultation with gynecology, urology, and general or colorectal surgery is preferred, but the general surgeon may not always have this luxury if their colleagues encounter unexpected findings at operation.^{44, 46} A variety of surgical techniques can be employed, including coagulation or laser ablation of implants, and the use of monopolar or bipolar electrocautery and harmonic scalpel. Lysis of adhesions should be performed when possible. Endometriomas should be removed in their entirety, as this procedure has been demonstrated to have a lower recurrence rate and greater improvement in pain when compared to drainage alone. Radical surgery, including hysterectomy and bilateral salpingo-oophorectomy should be reserved for women with severe disease and in whom future fertility is not an option. Laparoscopic uterine nerve ablation (LUNA) has been performed for endometriosis-related pain, but is not currently recommended. Presacral neurectomy, which employs chemical neurolysis of the hypogastric

plexus, has been shown to improve symptoms in those with midline pain, but should not be performed routinely. The use of postoperative hormonal suppression therapy is controversial.^{42, 44, 45, 47-49} In stage IV cases, large or small bowel resection, bladder resection, colostomy, and other procedures may be necessary.

The treatment of diagnosed endometriosis in the non-emergent setting involves minimizing the complications of endometriosis, including pain and infertility, and should be managed by a gynecologist. The management of endometriosis-associated pain may be medical or surgical. Medical management consists of hormonal suppression. In general, all medical options for the management of endometriosis-related pain have been shown to be effective, with none providing superior relief of pain. Surgical management should be reserved for cases of intractable pain after medical management and complications of endometriosis.

CT and MRI are infrequently used in the diagnosis of endometriosis, although these modalities may be helpful in differentiating between causes of acute abdominal pain in female patients. On CT scan, an endometrioma may appear as a cystic mass with hyperdense clot within. CT scanning may be more helpful with the presence of multiple or bilateral lesions or when endometrial implants are present in unusual locations. MRI is superior in the detection of blood within endometriomas, which is visualized as a hyperintense signal on T1-weighted imaging or hypodense signal on T2-weighted imaging. Small pelvic implants and pelvic adhesions may be better visualized on MRI compared to CT or ultrasound.^{2, 30} Double-contrast barium enema may be superior to MRI in the determination of enteric involvement of endometriosis and in preoperative planning in patients with bowel involvement.⁴⁶

Pelvic ultrasound may be helpful in the diagnosis of pelvic endometriosis, and specifically, endometriosis with ovarian involvement (endometrioma). On ultrasound, an endometrioma typically appears as a hypoechoic or anechoic cystic structure with posterior acoustic enhancement. Septations may be present, although nodularity should raise the suspicion for malignancy rather than endometrioma. The presence of peripheral, punctuate foci within the cyst wall is highly specific for endometrioma.^{2, 30} Ultrasound may be helpful when endometrial implants involve the urinary tract.⁴⁶

Catamenial pneumothorax is an interesting complication of endometriosis that may be encountered by the general or thoracic surgeon. It is defined as recurrent pneumothorax that occurs within 72 hours of onset of menses. Patients present with symptoms of pneumothorax, including cough, chest pain, and shortness of breath. The majority of catamenial pneumothoraces are right-sided. According to Alifano et al., patients with catamenial pneumothorax experience a mean of three pneumothoraces prior to definitive treatment, with some patients having more than ten episodes before receiving treatment. Patients may also present with hemothorax, hemoptysis, or endometriotic lung nodules.³⁹

Catamenial pneumothorax is typically considered the manifestation of thoracic endometriosis. The etiology of thoracic endometriosis is not clear, although transdiaphragmatic lymphatic or vascular transplantation of endometrial tissue has been proposed. Catamenial pneumothorax can be confirmed visually and histologically by the presence of endometrial glands and stroma within the pleura or diaphragm (Figure 5). Staining for estrogen and progesterone receptors is positive. Brown nodules or “blueberry spots” may be seen, which correspond to hemorrhagic foci and hemosiderin-laden macrophages.³⁹

Management of catamenial pneumothorax is both medical and surgical. Alifano et al. report that medical management alone is associated with a high recurrence rate in patients with catamenial pneumothorax (50% and 60% at 6 and 12 months, respectively). However,

surgical intervention alone is associated with 6 and 12-month recurrence rates of 5% and 25%, respectively. Current recommendations for management of catamenial pneumothorax are for video-assisted thoracic surgery (VATS) with removal or ablation of implants and pleurodesis, followed by 6 months of hormonal treatment. With diaphragmatic involvement, resection and repair can be done using an endoscopic stapling device or via minithoracotomy. Bullous lesions and endometrial lesions of the pleura should be resected. Talc pleurodesis is recommended over mechanical abrasion. The use of hormonal therapy (either OCPs, Danazol, or GnRH agonists) allows for ovarian rest and resorption of any small remaining endometrial lesions postoperatively. With the combination of surgical and medical therapy, the recurrence rate of catamenial pneumothorax is close to 0%.³⁹

Ruptured or Hemorrhagic Ovarian Cyst (Figure 6)

Massive rupture or hemorrhage of an ovarian cyst can present as a surgical emergency requiring immediate intervention. At times, the definitive diagnosis may be unclear, and the patient is taken emergently to the operating room due to hemodynamic instability. The most commonly reported ruptured ovarian cysts are functional cysts, including corpus luteum cysts, follicular cysts, and corpus albicans cysts. Endometriomas and dermoid cysts may also rupture, but are less common and will not be discussed in this section.^{15, 50-55} Rupture of a corpus luteum or follicular cyst is a physiologic event occurring during the menstrual cycle, and these cysts are prone to rupture due to increased vascularity. Rupture of a follicular or corpus luteum cyst is the most common cause of spontaneous hemoperitoneum in a pregnant patient, after ectopic pregnancy.^{15, 52, 56, 57}

Hemorrhagic ovarian cysts are most common in females of reproductive age, with the mean age at diagnosis ranging from 27-30 years.^{50-52, 56, 57} Rupture may occur between days 20-26 of the menstrual cycle, or during the first trimester in pregnant women. In fact, women may be aware of a sensation of pain and release of peritoneal fluid associated with the physiologic rupture of a corpus luteum cyst during ovulation, which is termed *mittelschmerz*. A ruptured ovarian cyst most commonly occurs on the right side, and therefore may be difficult to distinguish from acute appendicitis or other gynecologic causes of abdominal pain.^{15, 52, 57}

Sudden onset, acute abdominal pain is the most frequent presenting symptom of a hemorrhagic or ruptured ovarian cyst. Vaginal bleeding, nausea, vomiting, weakness, syncope, and shoulder tenderness have also been reported. Signs of circulatory collapse, including hypotension, tachycardia, and decreased hematocrit, may be present when massive hemorrhage has occurred.^{15, 50-52, 55} Fever and leukocytosis are unreliable. On physical exam a palpable abdominal mass, rebound tenderness, and guarding may be present. β -HCG testing should be negative.^{15, 30, 50, 52}

The differential diagnosis of a hemorrhagic ovarian cyst includes ectopic pregnancy, tubo-ovarian abscess, endometrioma, nonruptured ovarian cyst, adnexal torsion, dysfunctional uterine bleeding, gastroenteritis, or appendicitis.^{51, 55} As the diagnosis of hemorrhagic ovarian cyst may not be clear, ultrasound, either transabdominal or transvaginal, is the imaging modality of choice (Figure 6). Early in the course of a hemorrhagic ovarian cyst, the cyst may appear anechoic with a thin wall and posterior enhancement. As the blood begins to clot, internal echoes may appear, with a reticular, "fish-net" or "spider web" appearance. Fluid-fluid or fluid-debris levels may be present within the cyst. As the thrombus coalesces, discrete, rounded, hyperechoic, avascular masses may be visualized. On Doppler interrogation, the mass should be avascular, or a peripheral "ring of fire" flow may confirm the diagnosis of hemorrhagic ovarian cyst.^{2, 15, 30, 50, 51} Thrombi may be distinguished from loops of bowel by the lack of peristalsis and vascularity.^{30, 51} In the case

of massive hemorrhage, free intraperitoneal fluid may be present, while the cyst itself may be collapsed.^{2, 30, 51, 52}

CT scanning may also be employed when the diagnosis is unclear. On CT imaging an adnexal mass may be present with an internal fluid-fluid level. Hemoperitoneum is present as high attenuation fluid in the pelvis.^{2, 51} Compared to ruptured endometrial cysts, ruptured functional ovarian cysts (corpus luteum or follicular cysts) are smaller in diameter and have a smaller wall thickness, and are less likely to be bilateral and multilocular. Ruptured functional cysts are more likely to have a disrupted cyst wall, demonstrate active extravasation of contrast, and be associated with intraabdominal fluid.^{56, 57} MRI may be performed in cases in which ultrasound and CT are equivocal. A hemorrhagic ovarian cyst will have intermediate to high signal intensity on T2-weighted images, while any associated hemoperitoneum will be bright. Fluid-fluid levels may also be present due to thrombus and debris.²

The treatment of hemorrhagic ovarian cysts is dependent on the patient's presentation. Patients who are hemodynamically stable can be managed conservatively with analgesia and observation. Patients who present with hemodynamic instability should undergo emergent surgical intervention, even when the diagnosis is uncertain. When active and uncontrollable hemorrhage is present, oophorectomy should be performed. Otherwise, conservative management with preservation of the ovary should be attempted, especially in patients who desire future fertility.

When a ruptured ovarian cyst is encountered during a diagnostic laparoscopy for acute abdominal pain, if the cyst is small and there is no active hemorrhage, oophorectomy is not indicated. A careful examination for other pathology to explain the abdominal pain should ensue. If negative, conservative management with pain control is indicated. Copious irrigation of the pelvis should be performed.

Surgical intervention is also indicated for patients who do not respond to conservative management (>48 hours), or in whom symptoms and signs of worsening hemorrhage are present. Laparoscopy is the preferred method, while laparotomy should be reserved for cases in which laparoscopy is unsafe or not feasible (possibly due to bilaterality, cyst size, or surgeon ability), or when malignancy is suspected.^{15, 53-55, 58}

Aspiration or fenestration of the ovarian cyst is not recommended, as it has been associated with low diagnostic utility and high recurrence rates (0-40%). Currently, ovarian cystectomy is preferred over oophorectomy in the management of ovarian cysts. Wedge resection and fulguration have also been reported. Copious irrigation of the pelvis should be performed in all cases. Repeat ultrasound should be performed six weeks postoperatively to evaluate for resolution of the cyst.^{15, 52, 55}

Adnexal Torsion (Figure 7)

Adnexal torsion represents approximately 3% of gynecological emergencies.⁵⁹⁻⁶¹ Adnexal torsion is defined as a partial or complete twisting of the uterine adnexa around its vascular pedicle, including the infundibulopelvic ligament and tubo-ovarian ligament. The ovary, fallopian tube, or both may be involved.^{30, 60, 62} Vascular and lymphatic obstruction result, which may lead to arterial compromise and ovarian necrosis.³⁰ The right adnexa is most commonly involved and therefore may mimic appendicitis.^{30, 59, 61} This may be due to the longer utero-ovarian ligament on the right and the resulting hypermobility. The left adnexa has decreased mobility due to the presence of the sigmoid colon.⁶¹

Adnexal torsion can occur at any age, but the mean age is 32 years.^{60, 63} In adults, adnexal torsion is commonly associated with an ovarian mass, either a cyst or neoplasm, which provides a fixed point around which the adnexa may twist.² Adnexal torsion in children and adolescents is thought to be due to increased mobility of the adnexa.^{2, 30} Patients who have undergone previous pelvic surgery are at increased risk for adnexal torsion, with studies reporting 32-40% of patients with adnexal torsion having had previous pelvic surgery (tubal ligation, cystectomy, hysterectomy).^{60, 63} This may be due to the presence of postsurgical adhesions, around which the adnexa may torse.⁶⁰ It has been proposed that ovarian malignancy may cause adhesions or invasion of surrounding tissues, and may actually result in a decreased risk of torsion.^{30, 61} Ovarian hyperstimulation results in an increase in the size and weight of the ovary, which may be the mechanism by which these patients are at increased risk for torsion.⁶¹ Women who have undergone assisted reproductive technology are also at increased risk for adnexal torsion, which may be explained by the same mechanism as ovarian hyperstimulation.⁶⁴

The most common symptom of adnexal torsion is the acute onset of abdominal pain, typically in the lower quadrant.^{30, 59, 60, 63} A prolonged history of pain has been associated with a higher risk for ovarian necrosis.^{30, 61} A pelvic mass may be palpated in 47-97% of patients.^{59-61, 63} Other symptoms of adnexal torsion include nausea and vomiting, dysuria, and urinary retention, frequency, and urgency.^{59, 60, 63} Low-grade leukocytosis and fever may be present, but are less common.^{30, 59, 60, 63} Peritoneal signs may be present in extreme cases.^{59, 63}

The differential diagnosis of adnexal torsion includes appendicitis, pelvic inflammatory disease, tubo-ovarian abscess, ectopic pregnancy, adnexal or pelvic cyst, cholecystitis, and abdominal pain of unknown etiology.^{60, 63} A large percentage of patients with adnexal torsion have a delay in diagnosis due to failure to suspect the diagnosis early in the course.^{60, 63}

Ultrasound is the imaging modality of choice when adnexal torsion is suspected.^{2, 30} Typically, the torsed ovary will appear enlarged (>5cm in diameter) and edematous with echogenic areas of hemorrhage.^{2, 30, 62} Of note, women who have undergone assisted reproductive technology (ART) have larger cystic ovaries than untreated women.⁶⁴ Ovarian follicles and the uterus may be displaced laterally.^{2, 30} The “whirlpool sign” is considered to be the only direct sonographic finding of adnexal torsion.⁶¹ This sign is seen on Doppler ultrasound and is caused by twisting of the vascular pedicle around its axis, with the resulting image resembling a spiral or whirlpool.^{2, 30, 61} Absence of arterial and venous flow on Doppler ultrasound has been shown to be highly specific for adnexal torsion, and is associated with ovarian nonviability.^{2, 30, 59-61, 63} Free fluid may be seen in the pelvis when infarction and hemorrhage have occurred.^{2, 30}

When adnexal torsion is not suspected initially, a CT scan may be performed for other suspected diagnoses. An enlarged, displaced ovary may be visualized on CT scan. An associated mass may be seen, and the uterus may be shifted laterally. Other findings on CT scan include obliteration of surrounding fat planes and ascites. Surrounding hematoma or gas may be present if ovarian infarction has occurred. MRI may demonstrate vascular congestion or hemorrhage on T1-weighted images, in addition to a twisted vascular pedicle.²

The gold-standard therapy for adnexal torsion is surgery. In the acute setting, the general surgeon may be the only one available to perform the procedure. If adnexal torsion is encountered unexpectedly in laparotomy or laparoscopy for unclear diagnosis, the management is identical to that described below. The surgical treatment of adnexal torsion ranges from conservative, adnexa-sparing procedures to complete oophorectomy or

salpingectomy. Currently, the standard treatment is detorsion with salvage of the adnexa, typically by laparoscopy.^{59, 60, 63} A large literature review reported that ovarian function returned in 88-100% of patients who underwent adnexal detorsion alone for adnexa that appeared necrotic.⁶¹ Oophorectomy or adnexectomy should be reserved for cases of severe vascular compromise, necrosis, peritonitis, or an ovarian mass. TAH-BSO should be reserved for postmenopausal women.⁵⁹ In cases where oophorectomy or hysterectomy is considered, a gynecological surgeon should be consulted.⁶¹ An additional option for the surgical management of patients with adnexal torsion is ovariopexy. Huchon et al. report that ovariopexy should be performed in cases of malformation or excessive length of the utero-ovarian ligament in order to shorten the length of the ligament and reduce ovarian laxity.⁶¹

Operative times and postoperative hospital stays are shorter in patients undergoing laparoscopy, and patients who undergo laparoscopic treatment are less likely to have fever postoperatively.^{59, 63} Detorsion, either with or without ovarian cystectomy, oophorectomy, or salpingo-oophorectomy, is more common during laparoscopy, while total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO) is performed more often via laparotomy.^{59, 63}

If left untreated, torsion of the adnexa can result in ischemia and necrosis of the ovary, fallopian tube, or both. The resulting necrosis can lead to loss of the ovary, which may affect the patient's fertility.⁶² The risk of pelvic thrombophlebitis and thromboembolic complications with adnexal torsion is low, with a reported incidence of pulmonary embolism of 0.2%.^{61, 65}

Up to 25% of cases of adnexal torsion occur in pregnant women.² Adnexal torsion is the most common complication of an adnexal mass occurring during pregnancy, and typically occurs in the first and second trimesters.^{64, 66} Pregnant women are also at increased risk for recurrent adnexal torsion, either during the same pregnancy or subsequent pregnancies.⁶⁴ Compared to non-pregnant women with adnexal torsion, pregnant women with adnexal torsion are more likely to exhibit gastrointestinal symptoms and peritoneal signs on physical examination.⁶⁴ Ultrasound and MRI are the preferred methods of radiographic imaging during pregnancy due to the high sensitivity and low risk of radiation exposure to the developing fetus.¹⁹ However, Hasson et al. reported a 61% false negative rate when using the absence of vascular flow on Doppler ultrasound for the diagnosis of adnexal torsion during pregnancy.⁶⁴ Leukocytosis may be considered within normal range during pregnancy, and thus may be an unreliable sign.⁶⁴

In the case of adnexal torsion during pregnancy, surgical intervention should be performed emergently, regardless of gestational age.²⁰ Koo et al. reported the most frequently used laparoscopic procedures for adnexal torsion during pregnancy as salpingo-oophorectomy, cystectomy, and detorsion alone, with all procedures performed prior to 23 weeks gestation.⁶⁶ Cystectomy has been shown to be less common during pregnancy, while cyst fenestration is more common during pregnancy.⁶⁴ Ovariopexy may be performed in pregnant patients with recurrent episodes of adnexal torsion. However, if the torsion is caused by an ovarian cyst, cystectomy during the initial procedure can prevent subsequent episodes of torsion in this population.⁶⁴

Adnexal torsion in children is typically associated with increased mobility of the adnexa, although a pelvic or abdominal mass may be present.^{2, 30} Children typically present with symptoms similar to adults, with the abrupt onset of pain, nausea, and vomiting. There may be a delay in the diagnosis of adnexal torsion in children and adolescents, as symptoms may be attributed to other disease processes, including appendicitis.⁶⁷ Abdominal ultrasound is

the imaging procedure of choice in children. Laparoscopic surgery has been shown to be safe and effective in children, and is generally preferred.^{62, 67} Conservative management with untwisting of the adnexa is recommended as the initial step in the management of these patients, with subsequent oophorectomy depending on the appearance of the ovary after untwisting.⁶⁷ Oophorectomy is more common with a longer time to presentation and premenarche in the pediatric population.^{67, 68}

Uterine Fibroids (Figure 8)

Uterine fibroids, or leiomyomas, are the most common pelvic tumor in women. It is estimated that up to 25% of women of reproductive age have fibroids, although the overall prevalence has been reported to be as high as 75%.⁶⁹⁻⁷³ Uterine fibroids are more common in black women than in white women.^{72, 73}

Fibroids are benign tumors consisting of smooth muscle (myometrial) cells. Specifically, fibroids tend to be hormone-responsive, which may result in progression during pregnancy or with the use of oral contraceptives, and regression after menopause.^{2, 73} In addition, fibroids have been associated with high levels of epidermal growth factor (EGF), insulin-like growth factors (IGF), heparin-binding growth factors, transforming growth factor- β , and type III collagen.⁷³

Symptoms occur in 50% of women with uterine fibroids, with the majority presenting in the 4th decade of life. The most common symptoms of fibroids include abnormal vaginal bleeding and pelvic pain and pressure. Urinary symptoms such as urinary frequency or urgency may result from compression of the bladder by a large fibroid. Hydronephrosis may result from chronic urinary obstruction. Degeneration occurs when the fibroid enlarges rapidly, perhaps during pregnancy, and outgrows its blood supply; fibroid degeneration may be associated with acute abdominal pain. Women with fibroids may also have a history of repeated miscarriages, premature labor, placental abruption, and postpartum hemorrhage.^{2, 30, 69-72} The symptoms of fibroids are usually not acute and are easily diagnosed and managed by gynecologists.

The diagnosis of uterine fibroids is usually by ultrasound, either transabdominal or transvaginal. The typical appearance of a fibroid on ultrasound is that of a well-defined, hypoechoic mass arising from the myometrium. Acoustic shadowing may be present. Intramural involvement should be identified by ultrasound. In the case of fibroid degeneration, the mass may appear necrotic, with anechoic, irregular cystic spaces within. Saline instillation into the uterine cavity may help delineate fibroids on sonography.^{2, 30, 70} Uterine fibroids are a common incidental finding at laparotomy or laparoscopy and do not require intervention by the general surgeon.

Acute complications of uterine fibroids are rare and include deep vein thrombosis with or without pulmonary embolism, acute urinary retention with or without renal failure, rupture with intraperitoneal hemorrhage, mesenteric vein thrombosis, acute degeneration, or torsion of the fibroid. In most cases, patients should be managed according to their presentation, with definitive treatment of the fibroid postponed until the patient has stabilized. In the case of hemorrhage, thrombosis, or torsion, the associated fibroid may only be discovered at emergency laparotomy.⁷¹ Figure 9 shows an MRI of a pedunculated, degenerative uterine fibroid in a 30 week pregnant patient presenting with right lower quadrant pain and leukocytosis. MRI images were obtained and the pedunculated fibroid was mistaken for a large appendix. The appendix was normal at laparotomy and the fibroid was identified. Appendectomy was performed and gynecology recommended leaving the uterine fibroid in place, as any operation of the myometrium during pregnancy could induce preterm labor.

The elective treatment of uterine fibroids can be medical or surgical. In particular, a variety of hormonal therapies have been used in the management of fibroids due to their hormone-responsiveness. Gonadotrophin-released hormone (GnRH) agonists downregulate circulating estrogen and progesterone, resulting in a reduction in uterine volume and bleeding and fibroid size. Administration of GnRH agonists produces a menopause-like state, with side effects such as hot flashes, vaginal dryness, headaches, depression, and osteoporosis. Add-back therapy has been advocated to help alleviate side effects, but the addition of low-dose estrogen or progesterone therapy may also diminish the effect of the GnRH agonist on fibroid size.⁷³

Antiandrogen therapies such as danazol and gestrinone have been shown to result in amenorrhoea and improvement in menorrhagia, pain, and uterine volume in women with fibroids. However, significant side effects include acne, seborrhea, arthralgias and myalgias, and may preclude their use in many women. Additional therapies that have been used in the medical management of uterine fibroids include both progesterone and antiprogesterone therapy, antifibrotic agents, selective estrogen-receptor modulators (SERMS), oral contraceptives, and non-steroidal anti-inflammatory drugs (NSAIDs).^{72, 73}

Historically, the surgical management for uterine fibroids consisted of laparotomy and hysterectomy. However, the issues of operative morbidity and future fertility have brought the utility of hysterectomy into question. The most minimally invasive surgical procedure is now myomectomy via hysteroscopy. Laparoscopy may be used to perform myomectomy or hysterectomy in women with symptomatic fibroids. Typically, laparoscopic myomectomy can be performed for a solitary fibroid less than 15 cm in size, or no more than three intramural fibroids of 5 cm in size.

Laparoscopic hysterectomy may be performed for women with fibroids and a uterus less than 16 weeks gestational size. Partial (supracervical) hysterectomy can be performed in the case of uterine fibroids, where there is no cervical pathology, although total hysterectomy eliminates the possibility of cervical disease in the future.⁶⁹ Other procedures that may be performed laparoscopically for the treatment of uterine fibroids include thermolysis, cryomyolysis, and uterine artery ligation.^{69, 70} Laparotomy may be necessary in women with large or multiple fibroids not amenable to laparoscopy.

With regards to the management of uterine fibroids by the general surgeon, intervention should be reserved for cases of acute complications of fibroids, including torsion, infarct, or rupture, in which the diagnosis is not made preoperatively. When encountered incidentally during abdominal surgery, management should be deferred to a gynecologic surgeon.

GYNECOLOGIC PATHOLOGY FOUND DURING PREGNANCY

The diagnoses mentioned above can certainly occur during pregnancy, and a positive β -HCG should not eliminate these conditions from the differential diagnosis. In particular, ovarian cyst rupture and adnexal torsion can frequently occur and are often misdiagnosed during pregnancy. In addition, physicians should not forget that typical general surgical problems such as appendicitis, acute cholecystitis, diverticulitis, and other causes of peritonitis, can occur during pregnancy. The diagnosis may be complicated by physiologic and anatomic changes during pregnancy; for example, the appendix may be displaced upward due to a gravid uterus and may be misdiagnosed as cholecystitis.

However, there are several distinct gynecologic causes of abdominal pain that should be carefully considered when a patient is found to be pregnant. These include ectopic pregnancy, chorioamnionitis, retroverted gravid uterus, spontaneous uterine rupture, and threatened abortion. Ectopic pregnancy can present with abdominal pain and hemodynamic

instability and may be encountered by the general surgeon, and will thus be discussed in detail here. The latter diagnoses are less common and are not typically seen by general surgeons when an adequate workup has been performed, and will therefore be discussed briefly.

Ectopic Pregnancy (Figure 10)

Ectopic pregnancy is any pregnancy that occurs outside the uterine cavity, and most commonly occurs in the fallopian tube (ampulla, isthmus, or fimbria), abdominal cavity, ovary, cervix (Figure 10A), and uterine cornua (Figure 10B).^{12, 30, 74} Ectopic pregnancy occurs in approximately 0.5-2% of diagnosed pregnancies, and is typically discovered between 6 and 10 weeks gestation. Ectopic pregnancy is currently the leading cause of death during the first and second trimesters of pregnancy, accounting for 10-15% of all deaths during this time.^{2, 14} Heterotopic pregnancy is defined as an intrauterine pregnancy and an ectopic pregnancy occurring simultaneously. The rate of heterotopic pregnancy is low, but is more frequent in women undergoing fertility treatments or in vitro fertilization.¹²

Risk factors for ectopic pregnancy include a previous ectopic pregnancy, previous pelvic surgery, history of PID, intrauterine device (IUD) use, diethylstilbestrol (DES) exposure in utero, smoking, and infertility.^{14, 30, 74} A recent study demonstrated that IUD use was not associated with an increase in ruptured ectopic pregnancy or the need for emergency laparotomy or blood transfusion.⁷⁵ More than 40% of women with ectopic pregnancy have no documented risk factors.^{12, 14}

The differential diagnosis of ectopic pregnancy includes miscarriage (threatened or complete), acute appendicitis, adnexal torsion, PID, TOA, nephrolithiasis, and ruptured corpus luteum cyst or follicle.⁷⁴ Because of the vague symptoms, up to 40% of ectopic pregnancies are misdiagnosed in the emergency department.⁷⁶

Symptoms of ectopic pregnancy include abdominal pain and vaginal bleeding.^{3, 12, 14, 30, 74} Location of the abdominal pain varies with the location of the ectopic pregnancy and may include the midline, lower quadrants, or both. Vaginal bleeding may range from scant to profuse, but may also be present in patients with an abnormal intrauterine pregnancy or miscarriage.¹²

On physical examination, an enlarged uterus or adnexal mass may be palpated. Abdominal or pelvic tenderness and cervical motion tenderness on speculum exam may be elicited. However, these signs are insensitive and approximately 10% of patients with an ectopic pregnancy have a normal pelvic exam. In a pregnant woman with abdominal pain and vaginal bleeding, the risk of ectopic pregnancy is 39%; with any risk factors, the likelihood increases to 54%. In particular, tachycardia, hypotension, and significant abdominal tenderness should raise the suspicion of a ruptured ectopic pregnancy. Physicians should suspect ectopic pregnancy in a pregnant woman in her first or second trimester who presents with abdominal pain and vaginal bleeding; however, no combination of clinical or laboratory findings is completely accurate in diagnosing ectopic pregnancy.^{3, 12, 14, 74}

Laboratory values have been employed to aid in the diagnosis of ectopic pregnancy. The initial test that should be obtained is a β -HCG level. With a negative serum β -HCG, the chance of pregnancy of any type is low.¹² A β -HCG level termed the discriminatory zone has been established, at which an intrauterine pregnancy should be visualized on ultrasound. This level is typically 4000-6500 mIU/mL for transabdominal ultrasound and 1000-2000 mIU/mL for transvaginal ultrasound.⁷⁶ Ectopic pregnancy should be suspected if β -HCG levels are above the discriminatory zone and an intrauterine gestational sac is not visualized on ultrasound. The levels are not concrete, however, as both ruptured and unruptured ectopic

pregnancies have been demonstrated in patients with β -HCG levels below 100 mIU/mL and above 50,000 mIU/mL.^{12, 74}

Transvaginal ultrasound has been shown to be superior to transabdominal ultrasound, and should be obtained regardless of β -HCG levels in patients in whom ectopic pregnancy is suspected.⁷⁶ The first step in ultrasonographic evaluation is the determination of the presence of an intrauterine pregnancy. In a normal pregnancy, a gestational sac should be visualized at approximately 5 weeks gestation, which corresponds to β -HCG levels of 1000-2000 mIU/mL.^{2, 12, 30} If no viable intrauterine pregnancy is visualized on ultrasound, the differential diagnosis includes ectopic pregnancy, miscarriage, or intrauterine pregnancy less than 5 weeks gestation.³⁰

The most specific findings of ectopic pregnancy on ultrasound are the presence of a gestational sac or fetal pole with cardiac activity outside the uterine cavity.³⁰ Other ultrasound findings that are suggestive of ectopic pregnancy include an adnexal mass that is separate from the ovary with associated empty uterus, free fluid in the pelvis, and a “tubal ring sign.” The “tubal ring” appears as a ring-shaped structure with an anechoic and vascular wall, typically between the uterus and ovary.^{2, 12, 14, 30, 74, 76} An endometrial stripe less than 8 mm thick is also suggestive of ectopic pregnancy.¹² In summary, patients who are HCG positive with an empty uterus and a pelvic fluid collection should be suspected of having ectopic pregnancy. The combination of serial β -HCG levels and transvaginal ultrasound has a sensitivity of 96% and specificity of 97% in the diagnosis of ectopic pregnancy.^{12, 74}

The general surgeon will most likely encounter the hemodynamically unstable patient with a ruptured ectopic pregnancy, which may or may not be diagnosed. Surgical intervention should be performed immediately. Two large-bore intravenous lines should be placed, and blood transfusions may be necessary. Rho(D) immune globulin (Rhogam) should be administered to Rh-negative mothers.¹²

The goal of surgical management of ectopic pregnancy should be to preserve fertility, as the majority of these patients are young and desire future pregnancies. Historically, surgical management of ectopic pregnancy was laparotomy with salpingectomy. However, with the advent of laparoscopic surgery, treatment options for ectopic pregnancy have been modified. Laparoscopy can be used for both diagnosis and treatment in patients with ectopic pregnancy. Laparoscopy has been associated with lower costs, shorter hospital stays and operative times, less intraoperative blood loss, and less pain. Laparotomy may still be needed in certain patients or due to surgeon expertise, and the safety of the patient should not be compromised in order to pursue a less invasive technique.

Currently, laparoscopy with salpingostomy with preservation of the ovary is the preferred method of treatment. Salpingostomy includes removal of the gestational sac through a 1 cm-long incision in the wall of the fallopian tube, with sparing of the fallopian tube itself. Salpingostomy is less invasive than salpingectomy, and subsequent fertility and ectopic pregnancy rates have been shown to be comparable between the two procedures. Salpingectomy may be indicated in patients with uncontrollable bleeding, a severely damaged fallopian tube, in those with a large gestational sac (>5 cm), and in those with a history of ectopic pregnancy in the same tube. Oophorectomy may be indicated for ovarian pregnancies, while cornual resection or hysterectomy may be performed in the case of cornual pregnancies (Figure 10C). The failure rate of salpingostomy is 8%; surgical failures can be successfully managed with MTX therapy. β -HCG levels should be monitored weekly until undetectable.^{12, 14, 74}

The general surgeon is most likely to encounter the patient presenting with and ectopic pregnancy without awareness of their pregnancy, further complicating the diagnosis. For this

reason, it is critical that the general surgeon test for urine β -HCG and, if positive, follow it up with serum levels and document an intrauterine pregnancy.

Unruptured ectopic pregnancy can be treated medically or surgically and should be deferred to obstetrics/gynecology. Surgical management for unruptured ectopic pregnancy should be reserved for patients with severe symptoms or severe bleeding, those with high or rising β -HCG levels, and for those who have failed, refuse, or have contraindications to medical management.¹⁴ Surgical technique is similar to that for ruptured ectopic pregnancy, as discussed above. Medical management includes the administration of methotrexate (MTX), in either a single dose or multiple dose regimen. MTX inhibits DNA synthesis in trophoblastic cells by deactivating dihydrofolate reductase. Side effects of MTX therapy include bone marrow suppression, elevated liver enzymes, nausea, diarrhea, skin rash, alopecia, and stomatitis.^{14, 74} Contraindications to MTX therapy include renal or liver dysfunction, peptic ulcer disease, leukopenia or thrombocytopenia, pulmonary disease, or immunocompromise.^{12, 74} For both regimens, weekly transvaginal ultrasound should be performed. β -HCG levels should be monitored weekly until less than 15 mIU/mL. Overall success rates for single and multiple dose MTX therapy for ectopic pregnancy are 88% and 93%, respectively. Ectopic pregnancy is typically resolved within three to seven weeks after initiation of MTX therapy. β -HCG levels have been shown to return to baseline faster after surgery compared to MTX therapy.^{14, 74}

Chorioamnionitis

Chorioamnionitis occurs during pregnancy, and can occur at term or preterm. With careful diagnostic workup, chorioamnionitis should not be encountered by the general surgeon. However, surgeons may be consulted either when there is diagnostic uncertainty (possibly due to patient instability) or a gynecologist is unavailable.

Chorioamnionitis or intra-amniotic infection (IAI) is defined as infection of the amniotic fluid, placenta, and/or decidua. The clinical diagnosis of chorioamnionitis is based on the presence of maternal fever ($>38^{\circ}\text{F}$), in addition to at least one of the following: maternal leukocytosis ($>15,000$ cells/ mm^3), maternal tachycardia (>100 beats/min), fetal tachycardia (>160 beats/min), uterine tenderness, or foul odor of the amniotic fluid.^{77, 78}

Chorioamnionitis occurs due to inoculation of bacteria into the amniotic fluid, and usually occurs after rupture of the membranes during pregnancy. Risk factors for chorioamnionitis include multiple vaginal examinations or transcervical instrumentation (internal monitoring devices, cerclage placement), longer duration of labor, longer duration of ruptured membranes, presence of meconium, and nulliparity. Colonization of the vagina with group B streptococcus has also been associated with an increased risk of chorioamnionitis.^{77, 78}

Organisms implicated in chorioamnionitis include *Bacteroides* species, group B streptococcus, *Escherichia coli*, *Lactobacillus*, *Ureaplasma urealyticum*, *Candida* species, and *Haemophilus influenzae*. Microbes associated with bacterial vaginosis, including *Mycoplasma homini*, *Gardenerella vaginalis*, and other anaerobes have been cultured.⁷⁸

The diagnosis of chorioamnionitis is usually made clinically. Fever and maternal/fetal tachycardia, in addition to leukocytosis and rupture of the membranes, should raise the suspicion of chorioamnionitis.⁷⁸ At times, amniotic fluid may be sampled via amniocentesis, with the gold standard for diagnosis of chorioamnionitis being amniotic fluid culture. White blood cell count and interleukin-6 and glucose levels have also been used to increase the sensitivity of amniotic fluid markers in the diagnosis of chorioamnionitis. Blood cultures are not typically helpful in the diagnosis and treatment of chorioamnionitis.⁷⁷

Antibiotic therapy and delivery are recommended as the treatment for chorioamnionitis. Patients treated with antibiotics immediately upon diagnosis of chorioamnionitis have decreased neonatal sepsis and death, and decreased maternal postpartum fever and hospital stay compared to patients who have antibiotic therapy delayed until after delivery. Current recommendations for antibiotic therapy are parenteral ampicillin and gentamicin. Anaerobic coverage with clindamycin or metronidazole is recommended in those patients undergoing cesarean delivery. Cephalosporins and vancomycin may also be used. There is controversy over the postpartum continuation of antibiotics, with some authors recommending continuation of antibiotics after cesarean delivery due to greater tissue injury during the procedure and higher rates of postpartum endometritis. The diagnosis of chorioamnionitis does not mandate cesarean delivery; current indications for cesarean delivery should be followed regardless of the presence of chorioamnionitis.^{77, 78}

Maternal complications of chorioamnionitis include increased rates of cesarean delivery, uterine atony, and postpartum hemorrhage. With appropriately administered antibiotics, septic shock, coagulopathy, and respiratory distress can be avoided. After cesarean delivery, women with chorioamnionitis are at increased risk for surgical complications of hemorrhage, wound infection, intra-abdominal abscess, thrombophlebitis or thromboembolism, and endometritis. Neonatal complications are almost entirely limited to preterm deliveries and include pneumonia, sepsis, meningitis, enterocolitis, intra-cranial hemorrhage, and respiratory distress.^{77, 78}

Again, the general surgeon will not typically encounter a pregnant patient with chorioamnionitis if accurately diagnosed. However, surgical consultation may be obtained by gynecology if they suspect acute appendicitis or other intraabdominal emergency and the clinical picture is not definitive. The diagnosis and management of these patients should be completed by the gynecology team once a general surgical emergency is ruled out.

Retroverted gravid uterus

Retroverted uterus occurs when the axis of the body of the uterus is flexed posteriorly toward the sacrum, away from the normal anteverted position.⁷⁹ Approximately 15% of women have a retroverted uterus, although the uterus typically migrates anteriorly to the normal anatomic position during pregnancy. In cases where the uterus remains retroflexed during pregnancy, the uterus may become incarcerated in the pelvic cavity. As the retroverted uterus grows, the anterior wall of the uterus stretches and becomes thin, termed uterine sacculation. Risk factors for retroverted uterus include adhesions between the uterine fundus and pouch of Douglas (due to pelvic inflammatory disease, endometriosis, or previous abdominal surgery), uterine malformations (didelphys or bicornuate), uterine prolapse, and pelvic tumors.^{80, 81}

Symptoms of retroverted gravid uterus can be vague, which may lead to general surgical evaluation. Patients with retroverted uterus may complain of symptoms of pelvic fullness, including abdominal discomfort or tenderness, and back pain. Dysuria, cystitis, pyelonephritis, and urinary frequency, urgency, and retention may result from displacement of the bladder superiorly and anteriorly. Constipation may be present. The uterine fundus may fail to increase in size, with resultant intrauterine growth restriction.^{3, 80, 81}

On pelvic exam, a large mass may be felt in the cul-de-sac. The cervix is typically displaced anteriorly and may be difficult to locate.^{80, 81} Haylen et al. recommend transvaginal ultrasound with an empty bladder as an accurate method of diagnosis of retroverted gravid uterus.⁷⁹ MRI may also be useful in the diagnosis, on which a stretched cervix, thickened posterior uterine wall and thinned anterior wall may be visualized.⁸⁰

Prior to 20 weeks gestation, repositioning of the uterus should be considered. After 20 weeks gestation, complications such as preterm labor and premature rupture of membranes are more likely. Repositioning of the uterus may be done with or without general anesthesia, and involves bimanual pressure on the uterine fundus via the posterior fornix of the vagina. Laparoscopy or colonoscopy can be used to facilitate the procedure. Laparotomy should be reserved for extreme cases in which other interventions have failed, and should be performed by a gynecologic surgeon. Patients with retroverted gravid uterus should be delivered by cesarean section, and the surgeon should be sure to make the incision high in order to avoid injury to the bladder, cervix, or vagina.^{80, 81} These procedures should be deferred to a gynecologic surgeon whenever possible.

Spontaneous uterine rupture

Uterine rupture is an obstetric emergency, and may be encountered by the general surgeon in a hemodynamically unstable pregnant patient.^{3, 82, 83} Uterine rupture occurs when a complete tear through the entire thickness of the uterus occurs. Incomplete rupture or dehiscence of the uterus occurs when a surgical uterine scar opens but the visceral peritoneum remains intact, and is of minimal clinical significance.^{84, 85} Complete uterine rupture has been reported to occur in between 0.0003-0.06% of pregnancies. The mean age at diagnosis of uterine rupture ranges from 28-32 years. The majority of cases of uterine rupture occur after 28 weeks gestation.⁸²⁻⁸⁴

Risk factors for uterine rupture include previous cesarean section, multiparity, hypertension, oxytocin or prostaglandin use, preterm delivery, and malpresentation.⁸²⁻⁸⁴ Women with uterine rupture have higher rates of malpresentation, cephalopelvic disproportion, dystocia, non-reassuring fetal heart tones, and cesarean deliveries. Uterine rupture has also been associated with increase perinatal mortality, poor Apgar scores, postpartum hemorrhage requiring blood transfusion, and cervical tears.⁸³

Patients with uterine rupture will present with acute onset abdominal pain. Signs of shock, including maternal hypotension and tachycardia, and non-reassuring fetal heart tones may be present.^{3, 84, 85} Ultrasound may demonstrate a protruding amniotic sac, an endometrial or myometrial defect, extrauterine hematoma or pregnancy, and hemoperitoneum. MRI may identify an empty uterus with an extrauterine amniotic sac or pregnancy.⁸⁵

The surgical management of a ruptured uterus may in fact be performed by a general surgeon if a gynecological surgeon is not available. Delivery of the neonate is performed, and preservation of the uterus should be attempted. Suture repair of the uterine defect and ligation of the hypogastric artery has been proposed by some authors. Hysterectomy should be reserved for multiparous women not desiring future pregnancies, and in those with severe hemorrhage. Even after an episode of uterine rupture, women can still become pregnant with low maternal and fetal morbidity and mortality. Future deliveries should be by cesarean section.^{82, 84}

Similarly, uterine rupture may occur when a pregnant woman is involved in a traumatic accident, and may be encountered by the general surgeon at the time of laparotomy. The situation is rare and is associated with a fetal mortality rate approaching 100%. Ultrasound or CT imaging may be necessary, as pregnancy may mask the symptoms of hypovolemia typically experienced by trauma patients in severe shock. At the time of laparotomy, preservation of the uterus, as described above, can be performed. However, in the case of uncontrollable bleeding or severe shock, emergent hysterectomy may be needed.⁸⁶ An obstetrics/gynecology consult should be strongly considered when a pregnant patient is involved in a traumatic accident.

Threatened or spontaneous abortion

The diagnosis of threatened abortion is based on first trimester vaginal bleeding, documented fetal cardiac activity on ultrasound, and a closed cervix.⁸⁷ Lower back or abdominal pain may be present.³ Spontaneous abortion (miscarriage) occurs by 20 weeks gestation, with the majority of occurring by the 16th week.³⁰ This entity is only very rarely confused with a general surgical emergency and will not be commonly encountered by the general surgeon.

Risk factors for first trimester miscarriage include advanced maternal age, alcohol and drug abuse, caffeine use, cigarette smoking, maternal chronic diseases and infections, obesity, medications, reproductive tract abnormalities, and environmental toxins. Air travel, exercise, sexual activity, and stress have not been associated with an increased risk of miscarriage.⁸⁸

Patients with spontaneous abortion typically present with abdominal or pelvic pain and vaginal bleeding.³⁰ Size-date discrepancy on bimanual exam may be noted.⁸⁸ In both threatened and spontaneous abortion, β -HCG should be positive. Serial β -HCG levels can be monitored when the diagnosis is unclear.⁸⁸ Ultrasound can be used to differentiate between the two diagnoses. Fetal cardiac activity should be noted in the case of threatened abortion. With spontaneous abortion, the absence of cardiac activity (documented by 2 or more observers for 1-3 minutes) in an embryo with a crown-rump length of 5-6 mm should be noted. A complete abortion is defined as complete expulsion of the products of conception, and in this case, an empty uterus can be visualized on ultrasound.³⁰

Patients with a diagnosis of threatened abortion can be managed conservatively, and there is no role for the general surgeon in these patients. There is currently no documented benefit of uterine muscle relaxants, progesterone, or human chorionic gonadotropin in cases of threatened abortion.⁸⁸ Patients with threatened abortion have an increased risk of pregnancy-induced hypertension, pre-eclampsia, eclampsia, antepartum hemorrhage, placental abruption, preterm premature rupture of membranes, cesarean delivery, preterm delivery, low birth weight, and perinatal morbidity and mortality.^{87, 89} These women should be counseled appropriately.

Management of spontaneous abortion can be expectant, medical, or surgical, although a positive β -HCG level should lead to consultation of and management by the gynecology team. Expectant management may be chosen by some patients in an attempt to avoid intervention. Completion of the miscarriage occurs in up to 90% of patients, but may take as many as 4 weeks to occur. In patients who are stable and without signs of infection, expectant management can be performed after appropriate counseling.^{88, 90}

Medical management may be chosen by patients who are unwilling to wait for the miscarriage to complete naturally. Oral or vaginal misoprostol, a prostaglandin analogue, has been shown to be safe and effective. Patients may experience abdominal pain and vaginal bleeding within 6 hours of administration.^{88, 90}

Surgical management of spontaneous abortion should be performed by an experienced gynecologic surgeon. Surgical intervention should be based on patient preference and in those who have brisk or prolonged bleeding, decreasing hematocrit levels, and in those with signs of infection.⁸⁸ Dilation and curettage (D&C) is most commonly performed in the operating room as an outpatient procedure. Complications of D&C include incomplete evacuation, postprocedural infection, hemorrhage, and uterine perforation or cervical laceration.⁹⁰ Manual or electric vacuum aspiration of the uterus can be performed in the office, and may be less anxiety-inducing and more cost-effective for patients.^{88, 90} These procedures should be performed by physicians trained in the management of obstetric

complications; general surgeons are rarely involved in the care of patients with threatened or spontaneous abortion.

Women who have one previous miscarriage have been shown to be at risk for pre-eclampsia, preterm premature rupture of membranes, and preterm delivery. With two or more miscarriages, an additional risk of placental abruption and placenta previa is noted. Three or more miscarriages has been associated with an increased risk of congenital malformations in subsequent pregnancies.⁸⁹

GYNECOLOGIC PATHOLOGY FOUND INCIDENTALLY DURING ABDOMINAL SURGERY

At times, a gynecologic process is encountered incidentally during elective abdominal surgery for an unrelated reason. The patient may be asymptomatic or unaware of their gynecologic pathology, and the question arises whether definitive management should be performed at the time of discovery.

Primary Ovarian Neoplasms (Figure 11)

Primary neoplasms of the ovary occur in women around the time of menopause. The mean age at diagnosis of an ovarian neoplasm ranges from 48-64 years of age, with malignant tumors presenting between ages 54-63. The incidence of malignant ovarian tumors increases with age. More than half of women with ovarian neoplasms are postmenopausal.^{53, 91-95} Ovarian carcinoma is the 5th leading cause of cancer-related deaths in women, with an estimated 21,880 new cases and 13,850 deaths in 2010.⁹⁶

Ovarian neoplasms can be benign, borderline, or malignant, and are classified based on their embryologic tissue of origin (epithelial, germ cell, mesenchyme). Epithelial tumors of the ovary are the most common and include serous, mucinous, endometrioid, clear cell, transitional cell, squamous cell, mixed epithelial, and undifferentiated tumors. Of these, serous neoplasms are the most common ovarian tumors, and half of these are considered malignant (serous cystadenocarcinomas).^{53, 97, 98} In contrast, ten percent of mucinous neoplasms of the ovary are malignant. General surgeons may encounter pseudomyxoma peritonei or pseudomyxoma ovarii, which occurs when mucin extrudes from the cyst into the ovarian stroma.^{97, 98} Endometrioid tumors are almost always malignant and represent 10-15% of ovarian carcinomas. These tumors have been associated with endometriosis, endometrial hyperplasia, and endometrial carcinoma. Clear cell tumors and transitional cell carcinomas of the ovary may occur but are much less common.^{97, 98}

Germ cell tumors are the second most common type of ovarian neoplasm. Mature cystic teratomas are the most common benign ovarian tumor in women less than 45 years of age, and may be filled with sebaceous material, hair follicles, skin glands, muscle, bone, or teeth.^{53, 97, 99} Dysgerminomas are considered the female equivalent of testicular seminomas, and β -HCG levels may be increased in these patients. Endodermal sinus tumors, or yolk sac tumors, are malignant tumors of the ovary, and may be associated with increased levels of α -fetoprotein.⁹⁷ The most common sex cord-stromal tumors are granulosa cell tumors and Sertoli-Leydig cell tumors. Granulosa tumors are associated with hyperestrogenemia, while Sertoli-Leydig tumors can result in virilization.^{53, 97}

At times, general surgeons may encounter an incidental ovarian mass during laparotomy or laparoscopy. A gynecology consult should be considered. In a premenopausal patient with a suspicious ovarian mass, complete staging should be performed, including biopsy and frozen section of the mass, cytology of pelvic washings, evaluation of the pelvic nodes, resection of the para-aortic lymph nodes at the level of the ovarian vessels, and examination of the upper

abdomen with resection of the omentum.^{100, 101} Salpingo-oophorectomy will likely be needed. Discussion with the family should take place regarding unexpected oophorectomy, particularly in women of childbearing age. Total abdominal hysterectomy may be required in a second procedure. In a postmenopausal patient with a suspicious ovarian mass, oophorectomy of the abnormal side is preferred over biopsy. Total abdominal hysterectomy with bilateral salpingo-oophorectomy can be performed during the first procedure if the mass is malignant.

When a benign cyst is encountered by the general surgeon, no intervention is necessary if the patient is asymptomatic. If symptomatic and diagnosed during workup for pelvic pain, treatment should be referred to a gynecologist and be aimed at ovarian preservation, particularly in women who desire future fertility. Laparoscopic cystectomy is currently accepted for management of benign ovarian neoplasms. In women with bilateral involvement or a history of unilateral oophorectomy, cystectomy should be strongly advocated in order to preserve fertility. Adnexectomy or oophorectomy may be acceptable in postmenopausal women.

Additionally, general surgeons may identify an ovarian mass during the preoperative workup for an unrelated surgical diagnosis. Women with ovarian neoplasms typically present with symptoms of abdominal discomfort and ascites, which may be confused with a general surgical diagnosis. Dysfunctional vaginal bleeding may occur. Rarely, a pelvic mass is palpated.^{53, 102} Women with primary malignant ovarian tumors are more likely to have a CA-125 level greater than 35 IU/ml.^{53, 91} False positive levels of CA-125 are frequently encountered in premenopausal women, but an elevated level in a postmenopausal woman should raise the suspicion of malignancy.¹⁰³

Radiologic imaging can be helpful in the diagnosis and differentiation of ovarian tumors. Ultrasound is typically the first imaging modality used in evaluating a pelvic mass, although CT and MRI are commonly used. Benign tumors are more likely to be smaller, cystic, and have thin walls.^{53, 91, 97} Multilocularity, papillary projections, hemorrhage, and necrosis are more common in malignant tumors compared to benign tumors. Ascites, peritoneal implants, pelvic wall invasion, and pelvic lymphadenopathy may also be visualized.^{53, 91, 92, 97} CT scanning is particularly helpful in the detection of associated supraclavicular lymphadenopathy, pleural effusions, ascites, and peritoneal implants, and is therefore the imaging modality of choice for staging of ovarian carcinoma.^{95, 103} Figure 11A shows the CT scan from a female with abdominal pain and a dilated tubular structure near the cecum, ultimately metastatic ovarian carcinoma. The PET scan of the same patient in Figure 11B shows increased activity in the mass, omentum, and liver.

Image-guided biopsy has been suggested in cases of women with suspected advanced disease in whom major surgery could be avoided, in those with a history of malignancy, when the etiology of the primary tumor is unclear, and when the diagnosis is uncertain. Immunohistochemical staining may then be performed on the tissue sample. CA-125 and cytokeratin 7 stains may be positive in ovarian primaries, while carcinoembryonic antigen (CEA) and cytokeratin 20 stains are more likely in tumors of gastrointestinal origin. Image-guided biopsy may also allow for palliative paracentesis in select patients.¹⁰⁴

Patients with high-grade tumors, those with cyst rupture, and all patients with advanced stage disease (Stage IIIC or IV) are candidates for adjuvant chemotherapy. Recent studies have evaluated the use of neoadjuvant chemotherapy for the treatment of advanced or possibly unresectable ovarian carcinoma. Vergote et al. randomized patients to primary cytoreductive surgery followed by platinum-based chemotherapy or neoadjuvant platinum-based chemotherapy followed by interval debulking surgery and postoperative

chemotherapy. Stage IIIC disease, no residual disease after surgery, smaller tumor size, endometrioid type tumors, and younger age were associated with improved survival.⁹⁴

Ovarian Metastases (Figure 12)

The ovary is a common site of metastasis, with approximately 5-30% of ovarian malignancies being metastatic from another site. At times, the ovarian disease is recognized prior to, concurrent with, or after the diagnosis of the primary tumor, and may be encountered incidentally by the general surgeon. The exception to this is in breast cancer, where the primary breast carcinoma is almost universally identified prior to recognition of the ovarian metastases.^{91, 105-107} Tumors may spread to the ovary by a variety of routes, including lymphatics, blood, transperitoneally, or by direct extension from adjacent organs.¹⁰⁸

The mean age at diagnosis of a metastatic ovarian tumor ranges from 46-59 years. Patients may be pre- or postmenopausal.^{91, 92, 105-107} Approximately 60% of women with metastatic tumors to the ovary have a known history of cancer.^{91, 105} Up to 75% of secondary ovarian malignancies are from non-gynecologic sites, with the majority of these cases comprised of metastases from gastrointestinal (GI) tract and breast cancers. The most common GI tract malignancies metastatic to the ovary are the stomach, large intestine, small intestine, and appendix. Metastases from the breast, pancreas, gallbladder, peritoneum, and carcinoid tumors have also been reported. Gynecological malignancies have been reported to originate from the endometrium, cervix, and fallopian tubes. Other less common malignancies which metastasize to the ovary include malignant melanoma, gastrointestinal stromal tumors, lung cancer (most commonly small cell carcinoma) renal cell carcinoma (most commonly clear cell type), transitional cell carcinoma of the bladder, cervical carcinoma (squamous cell or adenocarcinoma), and endometrial carcinoma (stromal sarcoma, endometrioid type adenocarcinoma).^{91, 92, 105-110}

Krukenberg tumors are a specific type of metastatic tumors of the ovary, and are defined by the presence of mucin-filled signet-ring cells embedded in ovarian stroma. Approximately 75% of Krukenberg tumors originate from the stomach, although intestinal, breast, and biliary carcinomas have been reported to manifest as Krukenberg tumors as well. Mucinous tumors of the appendix, intestines, pancreas, gallbladder, stomach, cervix, and lung may metastasize to the ovary and be misdiagnosed as Krukenberg tumors.¹¹¹

When encountered intraoperatively, several pathologic features should point the surgeon toward the diagnosis disease metastatic to the ovary. In general, metastatic tumors to the ovary are more commonly bilateral when compared to primary ovarian malignancies, with up to 71% of cases involving both ovaries.^{105, 106} Secondary tumors of the ovary are typically smaller in size compared to primary tumors, with most tumors being 10 cm or less in diameter.^{91, 105, 106} Other features which should point the clinician toward a metastatic tumor of the ovary include a nodular appearance of the ovary, surface deposits of the tumor on the ovary, lymphovascular invasion, and an infiltrative pattern of stromal invasion. In addition, invasion of the ovary by a metastatic tumor can result in stromal luteinization and hyperandrogenic or estrogenic manifestations.^{108, 110}

Patients with tumors metastatic to the ovary typically present with abdominal complaints consistent with ovarian disease, but which may overlap with symptoms of other intraabdominal pathologies. These may include abdominal distention or increased girth and abdominal pain. Abnormal vaginal bleeding may result from endometrial hyperplasia due to excess estrogen production from the involved ovary. Patients with secondary ovarian tumors are less likely to have ascites compared to patients with primary ovarian malignancies, but are more likely to have carcinomatosis and intraabdominal adhesions.^{91, 105}

CA-125 levels may be elevated in patients with ovarian malignancies, although this value is not helpful in distinguishing between primary and secondary malignancies. However, Antila et al. noted that patients with metastatic tumors had higher levels of carcinoembryonic antigen (CEA).^{91, 105} Specifically, immunohistochemical staining in metastatic colorectal carcinoma is positive for CK20 and negative for CK7 and CA125, which may help distinguish these tumors from metastatic endometrial carcinoma.^{108, 110} Metastatic breast carcinoma stains positive for CK7 and negative for CK20. Estrogen and progesterone receptor staining is often positive. These tumors may also be diagnosed at the time of prophylactic oophorectomy.^{108, 110}

Tumors metastatic to the ovary may be visualized radiographically during workup of the primary malignancy. Secondary ovarian malignancies are less likely to be multilocular on ultrasound when compared to primary ovarian malignancies.⁹² CT scanning may be helpful in visualizing intraabdominal lymphadenopathy and additional metastases. Secondary ovarian tumors may demonstrate a soft tissue density on MRI, compared to primary ovarian tumors, which may have protein or bloody or clear fluid within. In general, it is difficult to distinguish between these types of tumors on radiologic imaging.⁹²

General surgeons may encounter a metastatic ovarian tumor before or during surgery for the primary malignancy. Figures 13A and B demonstrate a primary colon cancer and synchronous ovarian metastasis identified at the time of surgery. When metastatic disease of the ovary is identified intraoperatively, surgical resection should be performed if this is the only other site of disease. In premenopausal women, a discussion with the family should take place before performing unilateral or bilateral oophorectomy even if this requires leaving the operating room to do so. In addition, for many gastrointestinal tumors, the possibility can be mentioned during the preoperative consent process. In postmenopausal women, bilateral oophorectomy should be considered at the time of discovery. Lee et al. demonstrated that women who undergo extensive cytoreductive surgery have improved survival compared to patients who undergo incomplete removal of the metastatic tumor or biopsy only.¹⁰⁶ In patients with widespread carcinomatosis from a gastrointestinal primary, unilateral or bilateral oophorectomy is unlikely to be helpful.

Patients with metastatic tumors of the ovary have a poor prognosis overall. Patients with metastatic disease from a gynecologic primary have a better prognosis than those from a non-gynecologic primary, with a median survival of 24 months.¹⁰⁶ Two- and 5-year survival rates after surgical resection for a gynecologic primary have been shown to be 62% and 47%, respectively. The improved survival of patients with metastatic disease of gynecologic origin may be related to the surgical procedures which are typically performed for malignant gynecological tumors, including hysterectomy and salpingo-oophorectomy, which may remove the associated ovarian disease simultaneously.^{105, 107} Patients with metastatic disease from colorectal, gastric, and other non-gynecologic sites have median survival of 9, 9, and 12 months, respectively.¹⁰⁶ In patients with metastatic disease from a nongynecologic site, the overall 2- and 5-year survival rates are 46% and 19%, respectively. Patients with metastatic disease from the small intestine and pancreas have the shortest survival times.^{105, 107}

Bicornuate Uterus (Figures 13 and 14)

Bicornuate uterus is a congenital malformation that develops due to a failure of complete fusion of the Müllerian ducts during embryologic development. The Müllerian ducts (paramesonephric ducts) are evident between 6 and 7 weeks gestation, and develop along the urogenital ridge. These ducts elongate caudally and medially, and fuse to form the uterovaginal canal. Resorption of the intervening septum is completed by week 20 to form a uterus and upper vagina. Unification defects occur due to a defect in this developmental

cascade (Figure 11). Arcuate uterus is defined as a uterus with a small midline septum and a minimal fundal cavity indentation. Arcuate uterus is considered a normal variant and typically does not require intervention. A septate uterus is the most common Müllerian duct anomaly and develops when resorption of the intervening septum is incomplete. Bicornuate uterus develops due to failure of complete fusion of the Müllerian ducts and may be complete or partial. Two endometrial cavities and a single cervix and vagina result. Differentiation between septate and bicornuate uterus is crucial, as the management of these entities is distinct.¹¹²⁻¹¹⁶

Müllerian duct anomalies range in prevalence between 0.4-5.5% in fertile women and up to 38% of women with repeated pregnancy loss.^{113, 115-118} Specifically, bicornuate uterus accounts for up to 25% of Müllerian duct defects.^{118, 119} Bicornuate uterus has an estimated prevalence of 0.4% in the general population, 1.1% in infertile women, 2.1% in women who miscarry, and 4.7% in women who have struggled with both spontaneous abortion and infertility.¹¹³

Women with Müllerian duct anomalies may present with cyclical or noncyclical pelvic pain and dysmenorrhea. Endometriosis may also be present. In an extensive review, Rackow et al. suggest that Müllerian duct anomalies are associated with disrupted endometrial development and vascularity, diminished uterine size, musculature, and contractibility, and impaired cervical function. These factors may be associated with the increased rates of first and second trimester miscarriage, infertility, preterm delivery, malpresentation, intrauterine growth restriction, cervical incompetence, antepartum and postpartum bleeding, retained placenta, and cesarean deliveries seen in women with Müllerian duct anomalies.^{112, 116, 118, 119}

Diagnosis of bicornuate uterus can be performed by various imaging techniques. Both the internal anatomy and external surface of the uterus must be examined in order for an accurate diagnosis to be made. Imaging modalities include two-dimensional ultrasound, three-dimensional (3D) ultrasound, sonohysterography, hysterosalpinography, MRI, direct hysteroscopy, and laparoscopy. In patients with a bicornuate uterus, the urogenital system should be examined, as these women have a high rate of associated bladder, ureter, and renal defects (horseshoe kidney, renal agenesis, ectopic ureter).¹¹⁴⁻¹¹⁶

Typically, patients with bicornuate uterus do not present acutely, and therefore may not be seen emergently by the general surgeon. In rare cases, a bicornuate uterus can have an undrained horn. Figure 12 shows the bicornuate uterus of a 13 year old who presented with acute right lower quadrant abdominal pain at the onset of her menses. When encountered incidentally in asymptomatic patients during abdominal surgery, treatment should be postponed and the patient should be referred to a gynecologist. If identified for acute abdominal pain as in the case presented above, we would not recommend hysterectomy without intraoperative consultation from gynecology.

Surgical intervention is indicated in women with bicornuate uterus and significant pelvic pain, endometriosis, and poor pregnancy outcomes (repeated spontaneous abortions, infertility, preterm delivery). The goals of surgical intervention should be to restore normal pelvic and uterine anatomy and preserve or enhance fertility. Surgery should be avoided during the secretory phase of the menstrual cycle, when the vascular endometrium may cause excessive intraoperative bleeding. Historically, the Strassman's abdominal metroplasty was considered the treatment of choice for women with bicornuate uterus. However, this abdominal procedure was associated with high levels of postoperative pain, reduction of uterine volume, prolonged hospital stay, and an increased rate of pelvic adhesions, which may reduce fertility. In addition, abdominal metroplasty requires a waiting

period of 3-6 months before conception, although it has been shown to improve reproductive outcomes in women with bicornuate uterus. This can also be performed laparoscopically.

CONCLUSION

Female patients frequently present with acute abdominal pain, and although the signs and symptoms may be consistent with appendicitis and other general surgical emergencies, the diagnosis may, in fact, not be so clear. Specifically, a broad differential diagnosis should be kept in mind when faced with a woman of childbearing age with abdominal pain.

The initial step in managing a female patient with abdominal pain should be a complete history, including sexual, menstrual, and reproductive histories, and a physical exam, including pelvic examination. A β -HCG level should be drawn. Radiologic imaging, including ultrasound, CT, and MRI are commonly used.

In the non-pregnant female, appendicitis, pelvic inflammatory disease, tubo-ovarian abscess, endometriosis, ruptured or hemorrhagic ovarian cysts, and adnexal torsion can manifest with acute abdominal pain. Uterine fibroids may present acutely with torsion, hemorrhage, or infarct.

In a pregnant female, additional diagnoses can be encountered, including ectopic pregnancy, chorioamnionitis, retroverted gravid uterus, spontaneous or traumatic uterine rupture, and threatened or spontaneous abortion. Additionally, both pregnant and non-pregnant females may present with general surgical emergencies, including cholecystitis, diverticulitis, and peptic ulcer disease. Diagnosis and management of these disorders may be complicated in pregnant patients, and timely recognition of both the pregnancy and the cause of abdominal pain is key to maximizing outcomes for both the mother and fetus.

In addition, gynecologic diseases may be encountered incidentally by the general surgeon during abdominal surgery for an unrelated reason. These may include uncomplicated fibroids, bicornuate uterus, ovarian neoplasms, and ovary metastases. Typically, management of these disorders should be postponed and the patient referred to a specialist.

In summary, causes of acute abdominal pain are varied, and the diagnosis is not as clear as it may seem, particularly in female patients. A systematic approach to working up these patients can lead to a timely and accurate diagnosis and appropriate management.

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Figure 1.

(A) A CT scan of a 24-year-old female with fever, nausea, vomiting, and abdominal pain. An enlarged, fluid-filled tubular structure is noted in the pelvis, remote from the cecum. (B) More distal cut shows a right adnexal fluid collection and a left ovarian cyst (arrows).

Figure 2.
Laparoscopic view of perihepatic adhesions consistent with Fitz-Hugh-Curtis Syndrome.
(Source: http://commons.wikimedia.org/wiki/File:Perihepatic_adhesions.jpg.) (Color version of figure is available online.)

Figure 3.

Laparoscopic view of pelvic inflammatory disease with pyosalpinx (arrows). U, uterus.

(Reprinted with permission from Chandra S. ROLE OF LAPAROSCOPY IN THE MANAGEMENT OF PELVIC INFLAMATION DISEASE/TUBO-OVARIAN ABSCESS COMPARE TO OTHER MODALITIES. Available at:

<http://www.laparoscopyhospital.com/role-of-laparoscopy-in-the-management-of-pelvic-inflammation-disease-tubo-ovarian-abscess-compare-to-other-modalities.html>.) (Color version of figure is available online.)

Figure 4. Endometrial implants on the serosa of the terminal ileum. (Reprinted with permission from Chaer R, Sam A 2nd, Teresi M, Cintron J. Endometriosis-induced acute small and large bowel obstruction: rare clinical entities. *N Z Med J* 2005;118:U1521. (Color version of figure is available online.)

Figure 5.

A: Thoracoscopic view of endometriosis of the diaphragm in a patient with catamenial pneumothorax. B: Thoracoscopic view of endometriosis of the diaphragm in the patient's twin sister, also with catamenial pneumothorax. (Reprinted with permission from <http://catamenialpneumothorax.com/id15.htm>.) (Color version of figure is available online.)

Figure 6. Ultrasound demonstrating a hemorrhagic ovarian cyst. Internal septations and a reticular pattern appear within the cyst as the blood begins to clot.

Figure 7.
A: Intraoperative view of adnexal torsion. B: Intraoperative picture of a large, torted ovary.
(Color version of figure is available online.)

Figure 8.
Intraoperative picture of uterine fibroids. (Color version of figure is available online.)

Figure 9. T2-weighted coronal MRI demonstrating right lower quadrant fibroid (arrow) mimicking appendicitis during pregnancy.

Figure 10.

A: Laparoscopic view of cervical ectopic pregnancy. B: Laparoscopic view of cornual ectopic pregnancy. (Color version of figure is available online.)

Figure 11.

A: CT scan from a female patient with abdominal pain and a dilated structure near the cecum. The patient had previously had an appendectomy and was ultimately diagnosed with metastatic ovarian carcinoma. B: PET scan from the same patient, showing increased activity in the ovarian mass, liver, and omentum. (Color version of figure is available online.)

Figure 12.

A: Intraoperative view of primary adenocarcinoma of the colon (arrow). B: Intraoperative view of ovarian metastases from adenocarcinoma of the colon in the same patient. Bilateral oophorectomy was performed. C: Bilobed ovarian mass after oophorectomy for ovarian metastases from primary colon adenocarcinoma. D: Microscopic view of colon adenocarcinoma metastatic to the ovary. (Color version of figure is available online.)

Figure 13.

Spectrum of unification defects of the uterus. Arcuate uterus presents with small midline septum and a minimal fundal cavity indentation. A septate uterus is the most common Müllerian duct anomaly and develops when resorption of the intervening septum is incomplete. Bicornuate uterus develops due to failure of complete fusion of the Müllerian ducts; two endometrial cavities and a single cervix and vagina result. (Color version of figure is available online.)

Figure 14. Bicornuate uterus of a 13-year-old who presented with acute right lower quadrant abdominal pain at the onset of her menses due to an undrained uterine horn. (Color version of figure is available online.)

Table 1

Comparison of signs and symptoms of the differential diagnosis of acute appendicitis in females

	RLQ pain	LLQ pain	BLQ pain	Fever	Nausea/vomiting	Elevated WBC	Shock
Appendicitis*	+++	+/-	+/-	+	+/-	+	+/-
Pelvic Inflammatory Disease	+	+	+	+/-	+	+/-	+/-
Tubo-ovarian abscess	+/-	+/-	+/-	+/-	+	+/-	+/-
Endometriosis	+/-	+/-	+/-	-	-	-	-
Ovarian cyst	+/-	+/-	-	-	+/-	-	+/-
Adnexal torsion	+/-	+/-	-	-	+	-	rare
Complicated fibroids	+/-	+/-	+	-	-	-	rare
Ectopic pregnancy*	+/-	+/-	+/-	-	-	-	+/-

* Severe shock may result from sepsis due to appendicitis or hemorrhage due to ruptured ectopic pregnancy.