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Our public hospital emergency department (ED) has the "alley." Those of you of trained in public hospitals know what I mean: a place for ambulatory patients to be examined and treated, all the stretchers have stirrups, and the rooms are stocked with menstrual pads. You can often tell from the resident's facial expression that the next patient he or she is going to see is in the "alley." This area of the ED is supposed to be for more stable, less serious patients. However, over my years in emergency medicine, I've seen many patients deteriorate suddenly who were triaged to this area and I have learned to respect the "alley" and the patients who are seen there. This issue of Emergency Medicine Reports *is dedicated to that area, those patients, and the emergency physicians who care for them.*

—J. Stephan Stapczynski, MD, FACEP, FAAEM, Editor

Right Lower Quadrant Pain in Females

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Introduction

Abdominal pain in female patients can pose a diagnostic challenge to emergency physicians. There are a number of emergent clinical conditions that must be recognized in a timely fashion to reduce morbidity and mortality in these patients. This article will discuss these diagnoses and highlight critical points in the history and physical examination, as well as review current recommendations regarding treatment options and imaging modalities in this select group of patients. Both the pregnant and non-pregnant female patient will be discussed; however, traumatic causes of abdominal pain are beyond the scope of this review. Finally, miscellaneous causes of right lower quadrant pain that are not exclusive to females will also be discussed.

Emphasis is often placed on the initial history and physical examination of the patient, and with good reason. In most cases,

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the correct diagnosis can be established through a complete history, with careful attention to the gynecologic, obstetric, and sexual history. It should be noted, however, that patients often are reluctant to disclose their true sexual habits due to social stigmas. This is especially true among the lesbian, gay, bisexual, and transgender populations. It has been argued that self reports of sexual behavior are inherently unreliable and invalid due to multiple sources of bias, including underreports of stigmatized behavior and over reports of normative behavior.[1] Salient historical features that should be asked and documented in the chart include presence of an intrauterine device (IUD), history of prior sexually transmitted diseases (STDs), and specific factors associated with pelvic inflammatory disease (PID). These factors include age younger than 25 years, age of first intercourse at younger than 15 years, lower socio-economic status, single marital status, multiple partners, frequent vaginal douching, and self-reported history of *Chlamydia trachomatis*.[2]

When presented with a female of child-bearing age whose chief complaint is abdominal pain, it is essential to obtain a screening urine pregnancy test early in the evaluation. This will often determine the clinical/diagnostic pathway that should be pursued and can have implications in management options (e.g. choice of medications and radiographic procedures). The diagnostic algorithm provided can help the clinician approach the female patient with right lower quadrant pain.

RLQ Pain in the Non-pregnant Patient

Pelvic Inflammatory Disease (PID). Pelvic inflammatory disease must be considered as a diagnosis in all female patients presenting with lower abdominal pain (LAP). This disease entity comprises a spectrum of processes such as salpingitis, endometritis, and tubo-ovarian abscesses (TOA), all of which begin as an ascending infection originating in the cervix and vagina.[3]

Each year in the United States, more than 1 million women experience an episode of acute PID and an estimated more than 100,000 women will become infertile as a result.[4] A large proportion of this group is also at risk for subsequent ectopic pregnancies secondary to the adhesions and fibrosis that develop, which impede normal embryo migration through the fallopian tube.[4-6] Early diagnosis (or at least suspicion of) and the initiation of appropriate antibiotic therapy are the primary goals of the emergency physician.

Neisseria gonorrhoeae and *Chlamydia trachomatis* are the most frequent organisms isolated in patients with PID, although other microbes have been isolated. These include anaerobes, *Haemophilus influenza*, *Streptococcus agalactiae*, *Mycoplasma hominis*, and *Ureaplasma urealyticum*. Co-existent HIV infection has also been associated with increased progression to TOA.[7] This has been shown to be true regardless of CD4 T-lymphocyte count and is thought to be due to the impaired T-cell/macrophage signaling cascade and humoral mediated immunity. The causative agents in the HIV-positive population is also noteworthy. In a prospective observational study in which 162 female participants with lower abdominal were recruited, it was noted that HIV-positive women were less likely to be infected with either *N. gonorrhoeae* or *C. trachomatis*, or both, than were HIV-negative women.[8]

Clinical Presentation. The diagnosis of PID is principally based on clinical findings. The most common presenting complaint in patients with PID is lower abdominal pain that is often accompanied by a vaginal discharge.[9] Direct visualization of the fallopian tubes by laparoscopy is the current gold standard for the diagnosis of PID, but procedure is not routinely used in clinical practice.[10]

The clinical diagnostic criteria published in 2006 by the Centers for Disease Control and Prevention (CDC) are divided into "minimum criteria" and "additional criteria." (*See Table 1.*) Empiric treatment should be initiated in young sexually active women or those with risk factors associated with STDs who have one of the minimum criteria and where no other etiology can be identified. The additional criteria were added to enhance the diagnostic specificity and support a diagnosis of PID, although with reduced sensitivity.[4]

In one study, 189 patients diagnosed with PID by a gynecologist using clinical criteria were retrospectively studied in an attempt to describe the clinical basis for the diagnosis of PID and compare those to the CDC guidelines.[11] They found abdominal pain to be the most commonly encountered presenting symptom (98% of patients), followed by vaginal discharge (45%), feeling

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Table 1. Pelvic Inflammatory Disease: CDC Criteria (2006)

MINIMUM CRITERIA	ADDITIONAL CRITERIA
• Abdominal pain • Cervical motion tenderness • Adnexal tenderness	• Fever > 101°F (38.3°C) • Abnormal cervical/vaginal mucopurulent discharge • Elevated erythrocyte sedimentation rate and/or C-reactive protein • Presence of abundant numbers of white blood cells on saline microscopy of vaginal secretions • Positive cervical cultures with *N. gonorrhoeae* or *C. trachomatis*

Table 2. Intravenous Antibiotic Recommendations for PID, CDC

IV Regimen A	Cefotetan (Cefotan) 2 g IV every 12 hours **Or** Cefoxitin (Mefoxin) 2 g IV every 6 hours **Plus** Doxycycline 100 mg orally (preferred) or IV every 12 hours
IV Regimen B	Clindamycin (Cleocin) 900 mg IV every 8 hours **Plus** Gentamycin loading dose IV or IM (2 mg/kg of body weight), followed by maintenance dose (1.5 mg/kg) every 8 hours
Alternative IV Regimen (pen/ceph allergic)	Ampicillin/sulbactam (Unasyn) 3 g IV every 6 hours **Plus** Doxycycline 100 mg orally (preferred) or IV every 12 hours

of sickness (30%), dysuria (27%), and fevers and chills (25%). Examination of these patients revealed tenderness with manipulation of the cervix (cervical motion tenderness, or the Chandelier sign) and/or uterus and both adnexa in only 49% of patients.[11] As a result, the clinical diagnosis of PID is not a precise one, and considering the morbidity/mortality associated with the complications such as ectopic pregnancy and infertility, there is a generally accepted low threshold for initiation of treatment.

The distinction between acute appendicitis and pelvic inflammatory disease is an important one because it affects further evaluation and consultations in the ED. While both conditions may present with very similar signs and symptoms, there are a few noteworthy differences that should be sought during the initial history and physical examination. In a retrospective study of 181 women aged 12-58 who presented to the ED with abdominal pain, 3 clinical factors were more likely to be associated with a low risk of appendicitis: no pain migration (OR 4.2), presence of bilateral tenderness (OR 16.7), and absence of nausea or vomiting (OR 8.4). This study found that a prediction rule based on the presence of all three factors had a 99% sensitivity (95% CI 94-100%) for excluding acute appendicitis.[12]

Often, distinguishing PID from appendicitis on clinical grounds alone can be impossible, and imaging is required to accurately identify the cause of the patient's symptoms when appendicitis is suspected.

Diagnostic Imaging. The decision to image the abdomen in these patients should be pursued when alternative diagnoses/potential surgical disease (such as acute appendicitis, ovarian torsion, pelvic abscess) cannot be excluded from a non-surgical disease (ovarian cyst, PID.).[13]

Ultrasound is considered by some to be the most practical imaging modality in female patients presenting with lower abdominal pain. The advantages of ultrasound are that it is widely available, relatively inexpensive, has high diagnostic accuracy, and can be brought to the patient's bedside if needed.[14] Short of laparoscopy, pelvic ultrasound is considered the "gold standard" when making management and disposition decisions in patients with TOAs.[14] However, the ultimate accuracy of ultrasound is dependent on an experienced technician, a quality machine, and the skill of the interpreting radiologist, and some emergency departments may not have access to ultrasound imaging after hours and on weekends. In these cases, and in situations where appendicitis is a true consideration, CT should be pursued.

Focused appendiceal CT scans with oral contrast only (sensitivity 93%, specificity 97%, accuracy 96%) and thin (5 mm) cuts (sensitivity 99%, specificity 98%, accuracy 99%) have both been found to be useful in the distinction between appendicitis and TOA/PID.[15,16] When faced with the question of which imaging modality to begin with, one should use US if TOA is the suspected diagnosis and CT scan if appendicitis is the primary concern.

Treatment. The goal of PID treatment is to reduce the primary sequelae of chronic pelvic pain, ectopic pregnancy, and infertility. This results from the scarring and adhesion formation within the fallopian tubes as the inflammation resolves. Up to 18% of women treated for PID report chronic pelvic pain. The risk of infertility increases with each episode of PID: one episode is associated with a 13-21% risk, two episodes with a 35% risk, and three or more episodes with a 55-75% risk.[17]

The treatment options to be discussed in this article are based on the 2006 CDC recommendations for antibiotic use in PID. These guidelines were just updated in April 2007.[18] The updates no longer include the use of fluoroquinolones to treat gonorrhea, due to high rates of antibiotic resistance. In general, antibiotic regimens should be effective against *N. gonorrhoeae* and *C. trachomatis*. Since anaerobes have been isolated from the upper reproductive tract of women with PID, the CDC currently advises that treatment should include anaerobic coverage.[4] (*See Table 2.*)

Some experts have suggested that all patients with PID be hospitalized so that bed rest and supervised treatment with parenteral antibiotics can be initiated.[4] The current consensus, although based on limited clinical data, is that women with PID of mild or moderate severity be treated as outpatients. Suggested criteria for hospital admission include:[4]

Table 3. Outpatient Antibiotic Recommendations for PID, CDC

Ceftriaxone 250 mg IM as single dose
PLUS
Doxycycline 100 mg orally twice daily for 14 days (with or without metronidazole [Flagyl] 500 mg orally every 12 hours for 14 days)
OR
Cefoxitin 2 g IM in a single dose
AND
Probenecid 1 g orally administered concurrently in a single dose
PLUS
Doxycycline 100 mg orally every 12 hours for 14 days (with or without metronidazole [Flagyl] 500 mg orally every 12 hours)

- Surgical emergencies cannot be excluded after imaging
- Pregnant patient
- Lack of clinical response to oral antibiotic therapy after 72 hours
- Patient is unable to tolerate outpatient oral regimen
- Severe illness with nausea, vomiting, and/or high fever
- Presence of TOA
- HIV patients with low CD4 counts or other immunosuppressed patients (chronic steroid use, diabetic, etc.)
- Patients with intrauterine device (IUD)
- Consider admission for adolescent patients (unreliable compliance, increased risk for future ectopic).

Special consideration should be given to patients with concomitant HIV infection and low CD4 counts (< 350 cells/mm^3), as they are more likely to have complications and require admission. In one prospective study, 148 women with lower abdominal pain who were diagnosed with PID confirmed by laparoscopy (in which 39% were seropositive for HIV) there was no difference in clinical response to therapy in both seronegative and seropositive groups. However, there was a trend for increased risk of TOA in the seropositive group (2.8 fold increased odds), increased clinical severity scores, and longer hospitalizations. As opposed to the previously mentioned study by Bukusi, et al.,[8] the study by Cohen, et al.,[19] found no difference in microbiological etiology of PID comparing HIV-positive patients with those that were HIV negative.

If admission is deemed appropriate, the following parenteral regimen(s) should be initiated as soon as the diagnosis is made. (*See Table 2.*)

Patients who do not meet admission criteria and are clinically of mild to moderate severity may be discharged home with oral therapy. The current CDC recommendations (*see Table 3*) no longer recommend fluoroquinolones for the treatment of gonorrhea due to increasing resistance rates.

Clinical improvement should be seen within 3 days. If there is no response to therapy, an alternative diagnosis should be sought and/or the patient should be admitted for initiation of parenteral therapy. Male sex partners should be treated for presumed infection with agents effective against *C. trachomatis* and/or *N. gonorrhoeae*, due to the risk of re-infection of the patient. It is important to remember that these infections are often clinically silent in males, and the patient would otherwise not seek medical care. The presence of STDs is a significant risk factor for HIV infection, and all patients should be encouraged to have HIV testing performed when being treated for STDs.[4] The patient should be referred to the health department if HIV testing in the ED is unavailable.

Adnexal Torsion (AT). Although relatively uncommon in the ED, adnexal torsion represents the fifth most common surgical gynecologic emergency.[14] It is a surgical emergency because prompt operative detorsion is necessary to preserve ovarian viability.[20] This condition is most commonly seen in women of childbearing age, although it occurs in pre-menarche and post-menopausal women as well. Ovarian masses such as cysts (benign cystic teratomas or polycystic ovaries) or tumors increase the likelihood and risk of torsion[21] due to the increased size of the ovary. Increases in ovarian size > 6 cm predispose the patient for torsion. Thus, women undergoing hormone injection therapy for assisted reproduction have very high risk for torsion, due to the large number of cysts induced.[22] Risk of ovarian torsion also rises during the first trimester (6-14 weeks) and in the first 6-8 weeks after delivery, and 25% of cases are seen in pregnant patients.[23]

Adnexal torsion occurs because of the relative mobility of both supporting pedicles: the ovarian ligament connecting the ovary to the uterus and the suspensory ligament connecting the ovary to the intra-abdominal wall. During torsion, both pedicles are partially or completely strangulated, resulting in obstruction to venous outflow. This is soon followed by arterial flow obstruction. These events, if not corrected, lead to congestion, adnexal edema, and eventually ischemia and necrosis.[20]

Clinical Presentation. The typical presentation of a patient with a torsed ovary is usually described in texts as acute unilateral pelvic pain or a dull, aching pain with acute and sharp exacerbations if the torsion is intermittent. Case reports and large-scale reviews, however, have shown that the presenting signs and symptoms are usually less specific.[24] In fact, presenting symptoms of ovarian torsion are widely varied and can be very subtle. Thus, it is no surprise that many physicians fail to accurately diagnose this condition in a timely fashion, often because the physician did not consider this disorder in the differential of potential diagnoses.

In a published retrospective chart review, the average duration of pain lasted from several hours to weeks (mean 7.8 days). Thirty patients (35%) had mild tenderness on abdominal examination; 15 (17%) had severe tenderness. Twenty-five patients (29%) had no tenderness on pelvic examination, while 16 (18%) had severe tenderness.[24] Only 13 patients (16%) had a white blood cell (WBC) count greater than 15,000/mm^3, and most were afebrile and without nausea and vomiting. Interestingly, most of the diagnosed cases of adnexal torsion reported in the literature occur on the right side.[25]

The length of time required to render an ovary non-viable is unknown. The ability to retain viability despite prolonged ischemia as shown by preservation of ovarian function in even necrotic appearing ovaries indicates that complete arterial obstruction does not usually occur.[21]

Diagnostic Imaging. As with most gynecologic complaints,

pelvic ultrasound should be the initial imaging modality in suspected cases of adnexal torsion. Visualization of the ovaries and any adnexal masses, in addition to color flow Doppler imaging, make ultrasound the imaging modality of choice in the diagnosis of ovarian torsion.[14,26] However, in one study, normal Doppler flows were found in 60% of cases of ovarian torsion diagnosed by laparoscopy.[27] Thus, abnormal Doppler flows are highly predictive of AT, but a normal flow does not reliably exclude AT. This could be due to dual ovarian blood supply, intermittent nature of the torsion, and subtorsions. A case series demonstrated abnormally large ovaries in all patients with AT, a small amount of fluid in the cul-de-sac in 32%, and small cystic structures around the ovary (presumed to be follicles) in 74% of cases.[28] In the setting of an abnormally enlarged ovary, it is reasonable to consult a gynecologist, since this finding can be the only one in the setting of an AT.

Treatment. If the diagnosis is made or cannot be reasonably excluded, emergent consultation with a gynecologist should be undertaken, as definitive treatment is manual detorsion with or without ovarian resection.[20,21] In the meantime, the patient should receive analgesics, intravenous fluid resuscitation, and anti-emetics as needed.

Ruptured Ovarian Cyst/Ovarian Masses. Physiologic ovarian cysts are the most common gynecologic masses to occur in the female pelvis.[29] Ovarian cysts develop as a result of failure of a follicle to rupture or regress after completion of the follicular phase in the normal menstrual cycle. About two weeks into the normal menstrual cycle, the follicle ruptures and releases an ovum. This process can be a source of discomfort and is referred to as "mittelschmerz"—a German word translating as "middle pain," referring to the mid-cycle pain of ovulation. After release of an ovum, the follicle now involutes and becomes the corpus luteum. This structure will begin to produce progesterone, which will prepare the uterus for embryo implantation. The vast majority of ovarian cysts are physiologic in origin, unilocular, and non-neoplastic.[14]

Cystic structures identified on ultrasonic imaging are referred to as cysts if their diameter is equal to or greater than 2.5-3 cm. If they are less than this value, then they are simply referred to as follicles. During the course of the menstrual cycle, a mature graafian follicle or the corpus luteum ordinarily may reach a size of up to 2.5 cm. Therefore, the term ovarian cyst is reserved for structures larger than this size.[30]

Clinical Presentation. Most ovarian cysts are asymptomatic and may be discovered incidentally on a routine well-woman examination or encountered during a work-up in a female presenting with lower quadrant abdominal pain. When the cyst becomes symptomatic, it is typically due to cyst rupture or hemorrhage into the cyst. The most common presenting complaint is abdominal pain.[14,31] The pain can be sharp, intermittent, sudden in onset, and severe with associated nausea and vomiting. In some cases, cyst rupture can lead to life-threatening hemorrhage. Ovarian cysts also predispose patients to ovarian torsion.

Treatment. The sudden onset of abdominal pain with peritoneal findings would suggest ovarian cyst rupture, but the clinician should also consider an ectopic pregnancy and other causes of a surgical abdomen in this setting. Patients with symptomatic ovarian cysts should be referred to a gynecologist for outpatient work-up and follow up. All pre- and post-menopausal women with adnexal masses should have a prompt outpatient work-up for malignancy. The following ultrasound findings should raise the suspicion of an adnexal malignancy: masses larger than 7 cm; masses that persist beyond one menstrual cycle; and masses that have solid internal components or a complex cystic structure.[32] Warnings should be given to all, since there is always a risk of ovarian torsion or cyst rupture with hemorrhage.

RLQ Pain in the Pregnant Patient

Pregnant patients with right lower quadrant pain pose additional diagnostic challenges. In this patient population, it is important to consider both pregnancy-related and non-obstetrical etiologies of RLQ pain.

Relevant questions in the history include last menstrual period, previous STDs, previous or current IUD use, surgical history, previous pregnancies and any complications, presence or absence of vaginal spotting or discharge, and passage of tissue. Attempts should be made to quantify the amount of vaginal bleeding if present (ie., how many pads/tampons used in last 12-24 hours). Additionally, if the patient is aware of the pregnancy, a history of any prenatal visits is another important aspect of the history.

The physical examination should be guided by the patient's presentation and history and also by the gestational age. If bleeding after the 20th week of gestation is present, bimanual and speculum examination should be deferred until placenta previa is ruled out with an ultrasound evaluation. Like speculum examination, transvaginal US is NOT safe in a bleeding patient with suspected previa and should also be avoided.

Ectopic Pregnancy (EP). Accurately identifying ectopic pregnancy is essential in the patient who presents with abdominal pain in the first trimester, as EP is the most common cause of maternal death in this trimester.[33,34] The incidence of ectopic pregnancy in the United States has risen in recent decades to almost 2% of all pregnancies.[33] Risk factors for ectopic pregnancy include previous PID, previous or current IUD use, previous tubal surgeries, and previous ectopic pregnancies.[35] Additional factors associated with an increased frequency of ectopic pregnancy include: *in utero* diethylbestrol (DES) exposure, infertility, current smoking, three or more spontaneous abortions, induced abortions, and more than one lifetime sexual partner.[36]

Clinical Presentation. The "classic triad" of symptoms in ectopic pregnancy is amenorrhea, abdominal pain, and irregular vaginal bleeding.[37] However, less than 50% of patients with ectopic pregnancy will present with all three complaints.[36] Moreover, the above symptoms may also be found in an intrauterine pregnancy (viable or non-viable) as well as with cervicitis, PID, cervical irritation, or trauma.[36] Women may complain of nausea and/or vomiting, breast tenderness, and fatigue, symptoms that often occur with normal pregnancy. Physical examination findings of cervical motion tenderness, adnexal tenderness, and peritoneal signs may be present, but are neither sensitive nor specific for ectopic pregnancy.[33] An adnexal mass is palpable in only 10% of cases of ectopic pregnancy. Finally, a normal physical exami-

nation does not rule out an ectopic.[33,36]

Diagnostic Imaging. Transvaginal ultrasound is the current diagnostic modality of choice when ectopic pregnancy is suspected. With a high resolution ultrasound machine and a skilled ultrasonographer, intrauterine pregnancy (IUP) can be detected on transvaginal ultrasound with serum beta-HCG levels between 1000-1500 mIU/mL.[33,36] Variations in the discriminatory zone (lowest serum beta-HCG level at which a gestational sac can be visualized) are institution-dependent and related to the ultrasound equipment used and the skill of the ultrasonographer.[33] One should be aware of the discriminatory zone used at each hospital where one practices. In general, serum beta-HCG levels between 1000-1500 mIU/mL correlate with a gestational age of 4.5 to 5 weeks.[33] Multiple gestations are an exception in which an IUP may not be visualized at this discriminatory zone.

One important limitation of ultrasound is related to the location of the ectopic implant. Conventional 2-dimensional (2-D) US has variable sensitivity depending on the location of the ectopic. Interstitial pregnancy occurs with implantation of the gestational sac in the most proximal aspect of the fallopian tube. Interstitial pregnancies account for a small portion of ectopic pregnancies (2-6%) but carry an increased mortality risk due to the vasculature in the area, as there is increased potential for significant hemorrhage.[38] Seventy-one percent of interstitial pregnancies can be accurately diagnosed by ultrasound.[38] A cornual pregnancy is intrauterine by definition, with implantation occurring in the upper and lateral aspects of the uterus.[39] The distinction between interstitial and cornual implantations is therefore an important one, and if any ambiguity exists after 2-D ultrasound, obstetrical consultation is appropriate. Newer technologies such as 3-Dimensional and 4-Dimensional US may be superior to traditional ultrasound techniques in distinguishing interstitial from cornual implantations, although these modalities may not be readily available at many institutions.[38,39]

Definitive diagnosis of an ectopic pregnancy on ultrasound requires visualization of a gestational sac with yolk sac or fetal pole outside of the endometrial cavity. However, other ultrasound scenarios strongly suggest the diagnosis of ectopic pregnancy without providing a definitive diagnosis:

- An empty uterus with a beta-HCG level above the discriminatory zone;
- Presence of fluid in the cul-de-sac, especially if the fluid is echogenic or the amount of fluid is large;[40]
- Presence of an ill-defined adnexal mass.[33,34]

Any of the above findings should prompt obstetrical consultation for definitive diagnosis and further treatment.

Treatment. Patients with a confirmed early pregnancy (detection of beta-hCG in the urine or serum) who are hemodynamically unstable should be considered to have an ectopic pregnancy until proven otherwise. In these patients immediate resuscitation and obstetrical consultation are critical. The patient's clinical condition should guide fluid resuscitation and early transfusion, and transfusion should not be delayed awaiting consultation. Laboratory workup should include urinalysis, complete blood count, type and crossmatch, Rh status, coagulation panel, and a quantitative serum beta human chorionic gonadotropin (beta-HCG). The quantitative beta-HCG, while unimportant in the initial management of the unstable patient, will be useful for the consulting obstetrician in monitoring resolution of the ectopic pregnancy after intervention.[33]

Once an ectopic pregnancy is diagnosed or strongly suspected, further management decisions should be guided by an obstetrician. Treatment options for ectopic pregnancy in stable patients include medical therapy with methotrexate or emergent operative intervention. Although methotrexate has not received FDA approval for medical therapy in ectopic pregnancy, this treatment method has been endorsed by the American College of Obstetrics and Gynecology and is widely used at many institutions.[41] Treatment decisions depend on serum quantitative beta-HCG levels, gestational age, ectopic size, and presence or absence of fetal heartbeat. Importantly, 50 mcg of anti-Rho immunoglobulin should be given to all Rh-negative women presenting with an ectopic pregnancy or any vaginal bleeding in pregnancy.[33]

Heterotopic pregnancy is the presence of both an intrauterine pregnancy and an ectopic pregnancy. Estimates of the incidence of heterotopic pregnancy range from 1/2600 to 1/8000 in women who have not used assisted reproduction, and a reported incidence as high as 1-3% in patients using fertility drugs or in vitro fertilization.[34] In a woman without the aforementioned risk factors for ectopic pregnancy and signs and symptoms suggestive of an ectopic, this risk of heterotopic pregnancy is low and this condition can essentially be excluded *if* an IUP is visualized on ultrasound. Conversely, for patients with risk factors for ectopic pregnancy, the detection of an IUP by ultrasound does not eliminate the possibility of a heterotopic pregnancy. Therefore, depending on the risk for ectopic, consultation with a gynecologist would be prudent.

Miscarriage. Miscarriage should always be considered in the differential diagnosis of the pregnant patient with abdominal pain presenting in the first 20 weeks of gestation. Approximately 15% of pregnancies end in miscarriage, with most occurring before 12 weeks of gestation.[42] In addition to abdominal pain, vaginal bleeding is commonly present. The following definitions are useful in such scenarios:

Threatened miscarriage is defined as vaginal bleeding during the first 20 weeks gestation when fetal heart tones are present, without dilatation of the cervix.

Inevitable miscarriage is dilatation of the cervix with no history or evidence of passage of tissue in the first 20 weeks.

Incomplete miscarriage is passage of parts of the products of conception, in the presence of a closed cervical os, with retained fetal or placental tissue visualized on ultrasound.

Complete miscarriage is passage of all fetal tissue and subsequent closure of the cervical os before 20 weeks gestation.

Septic miscarriage is evidence of infection during any stage of miscarriage.[42]

Intrauterine fetal demise (IUFD) is defined death prior to the complete expulsion or extraction from its mother of a product of human conception, and further classified as early (< 20 weeks' gestation), intermediate (20-27 weeks' gestation), or late (> 28 weeks' gestation). To separate early pregnancy loss (spontaneous abortion)

from later pregnancy loss, a threshold gestational age (e.g., 20 weeks) or fetal weight (e.g., 350 g) is generally used. Most patients with IUFD report no pain, but may describe an absence of fetal movement. The diagnosis of IUFD is established by visualization of the fetal heart without any cardiac activity. Occasionally, patients with IUFD may present with abdominal pain and/or uterine bleeding. The available therapeutic options, including expectant, surgical, as well as medical management should be discussed with a gynecologist once IUFD is confirmed in the ED.

To evaluate vaginal bleeding and abdominal pain in the first 20 weeks, a pelvic examination should be performed to quantify the amount of vaginal bleeding, passage of tissue, and to examine for presence of cervical dilatation. Any tissue discovered in the vagina or cervical os should be sent for pathologic examination. Laboratory workup should include urinalysis, complete blood count, blood type, Rh factor and antibody screen, and a quantitative beta-HCG. Transvaginal ultrasound should be performed to evaluate for fetal viability, identify retained products of conception, and to evaluate for ectopic pregnancy.[42]

Treatment. Patients with a threatened miscarriage can be safely discharged home with warnings and instructions to follow up with an obstetrician in 2-3 days for repeat beta-HCG measurement.[42] Patients with the diagnosis of inevitable miscarriage or IUFD should have a gynecologist called to evaluate the patient in the ED; the discussion of treatment options and the risk of complications are best handled by this expert. Avoidance of tampons and sexual intercourse should be advised to prevent infection. If complete miscarriage is diagnosed, discharge with obstetric follow up is appropriate if vaginal bleeding is minimal.[42] If there is any concern for the possibility of retained fetal parts (incomplete miscarriage), an obstetrical consultation is appropriate, as these patients have the potential for a septic miscarriage and/or significant hemorrhage. The febrile patient with missed miscarriage should raise the concern for septic miscarriage, and mandates emergent obstetrical consultation, as well as blood and cervical cultures and Gram stain, intravenous antibiotics, and admission.[42] A commonly used antibiotic regimen in this scenario includes ampicillin (Omnipen) 1-2 grams IV q4-6 hours; gentamycin (Gentacidin, Garamycin) 3-5 mg/kg/day q6-8 hours adjusted for renal function; and metronidazole (Flagyl) 500 mg IV q8h or 750 mg IV q12h.[42] However, there are many other alternative regimens. Whichever antibiotic regimen is used, one must ensure adequate coverage for both aerobic and anaerobic organisms.

Administration of Rh immune globulin to Rh-negative women with first trimester spontaneous miscarriage is a common practice and supported by many expert opinions.[43] However, there are no studies that demonstrate that first trimester fetomaternal hemorrhage results in Rh sensitization.[43] From a risk/benefit standpoint, the administration of Rh immune globulin is a low-risk intervention, and could potentially lead to substantial benefits in future pregnancies. Dosing for Rh immune globulin is 50 mcg in the first 12 weeks of gestation and 300 mcg after 12 weeks.[42] If there is any doubt regarding gestational age, the 300 mcg dose should be administered.

Appendicitis In Pregnancy. The incidence of appendicitis in pregnancy is approximately 1 in 1500 pregnancies, a rate not different from non-gravid patients.[44] Numerous studies have reported a higher frequency of appendicitis in the second trimester as compared to the first and third trimester.[44,45]

Clinical Presentation. Early in pregnancy the presentation will not differ considerably from the non-gravid patient. However, differentiating between tubo-ovarian abscess, appendicitis, ectopic pregnancy, ovarian torsion, and ovarian cysts is impossible based solely on physical examination. Later in the pregnancy, the physical examination findings are often obscured, as the abdominal wall becomes stretched and the underlying inflamed appendix may not be in direct contact with the peritoneum.[34] Peritoneal signs may be absent as a result. Based on a small study from 1932, the classic teaching has been that abdominal pain from appendicitis may not be in the right lower quadrant as pregnancy progresses as the appendix is displaced upward by the growing uterus. One recent study disputes this long held belief.[46] A large retrospective study found that right lower quadrant pain was the most common presenting symptom in pregnant patients presenting with appendicitis regardless of gestational age.[47]

White blood cell count has limited utility in diagnosing appendicitis due to the physiologic leukocytosis of pregnancy. As with the non-gravid patient, a normal white blood cell count does not rule out appendicitis. Additionally, presence or absence of a left shift is not useful in predicting appendicitis.[44]

Diagnostic Imaging. The utility of ultrasound to diagnose appendicitis in pregnancy is often limited in the second or third trimester. The gravid uterus, obesity, the presence of a retrocecal appendix, and overlying bowel gas are all potential limitations when using ultrasound.[44] Studies have reported sensitivity as low as 75%, with specificities consistently above 95%.[34] Positive findings on ultrasound are reliable, while a negative or equivocal ultrasound study cannot reliably exclude appendicitis from the differential diagnosis. Thus, ultrasound has some value in the detection of acute appendicitis in the pregnant patient, but it also has real limitations.[48] Therefore, one should be aware that a normal US does not rule out appendicitis.

Just as with US, a CT scan that does not adequately visualize a normal appendix also does not rule out appendicitis. When abdominal ultrasound results are negative, equivocal, or ultrasound is unavailable, helical CT scan should be considered to rule out appendicitis. The diagnosis of patients who present early (< 12 hours) into their course of appendicitis remains a clinical one.

If CT scan is being considered it should first be discussed with a radiologist to select a protocol to minimize fetal radiation exposure. With a proper protocol selected to minimize fetal radiation exposure, helical CT scan of the abdomen involves a fetal radiation exposure of only 0.3 rads.[48,49] Diagnostic studies with fetal exposure of less than 5 rads are not believed to be associated with increased fetal risk of birth defects, growth retardation, or spontaneous abortion, regardless of trimester at the time the study is performed.[49] When helical CT scan or other imaging involving radiation exposure is indicated, the pregnant patient should be counseled regarding this information. This is consistent with guidelines set forth by the American College of Obstetricians and Gynecologists.[49]

One recent study using magnetic resonance imaging (MRI) to diagnose appendicitis in pregnancy shows promise, although further studies are necessary and this is not the current standard of care, primarily due to cost and limited availability.[50]

Treatment. Once appendiceal perforation occurs, both maternal and fetal mortality increase substantially. Maternal mortality rises to 4% with advanced gestation and perforation, while fetal mortality rises to as high as 20-25% after perforation has occurred.[51] Thus, early surgical consultation is appropriate when appendicitis is suspected or cannot be reliably excluded. It is also reasonable to discuss with the consultant the choice of diagnostic imaging in pregnancy, or the option of admission for serial examinations and observation. Additionally, parenteral antibiotics should be given early for suspected appendicitis, as this may reduce morbidity and mortality.[48,49] Penicillins and cephalosporins are considered to be safe in pregnancy—the exceptions being cefaclor and cephradine, which should be avoided.[49]

Once believed to be contraindicated during pregnancy, laparoscopic appendectomy has been used with increased frequency, particularly in the first trimester, with some institutions performing laparoscopic surgery in the second and third trimesters.[52,53]

Urolithiasis in Pregnancy. Kidney stones are a frequent cause of non-obstetric abdominal pain during pregnancy, with incidence estimates ranging from 1/1100 to 1/1500.[54,55] This does not differ substantially from rates seen in non-pregnant patients.[54] Elevated urine calcium levels and urinary stasis are believed to contribute to calculi formation in pregnancy. To counter this, ureteral dilatation and hydronephrosis naturally occur as the uterus becomes progressively enlarged, which may lead to fewer symptoms during pregnancy and easier passage of calculi.[54,55]

Clinical Presentation. The presentation of urolithiasis during pregnancy is similar to that of the non-pregnant patient: hematuria, nausea, vomiting, and flank or abdominal pain are the most common symptoms.[56]

The laboratory workup should be similar to the non-pregnant patient with suspected kidney stones. Serum creatinine levels are typically lower during pregnancy due to the increased glomerular filtration rate. The serum creatinine is of particular importance, as uremia increases the risk of fetal demise.

Diagnostic Imaging. Ultrasound should be used as a first-line diagnostic study, as it carries no potential risk of fetal harm from radiation.[34,55,56] Ultrasound will not show the stone but will show other features of the disease including hydroureter and hydronephrosis. The commonly used renal CT scan should be avoided when possible, especially during early pregnancy to prevent fetal exposure to unnecessary ionizing radiation.[34] Whenever ordering radiographic studies in a pregnant patient, it is useful to remember the guidelines set forth by the American College of Obstetrics and Gynecology:

1. Counseling that exposures < 5 rads have not been associated with increased fetal anomalies or spontaneous abortion.
2. Maternal health should not be compromised by fears of the dangers of radiation. However, alternative imaging procedures (such as US and MRI) should be employed whenever feasible.
3. Ultrasound and MRI are not associated with any known fetal adverse effects.
4. Radiologic consultation is advisable when necessary to estimate fetal radiation doses, especially when multiple radiographic tests are being performed.
5. Therapeutic radioactive iodine isotopes are contraindicated.
6. Radio-opaque and paramagnetic contrast agents are unlikely to cause harm, but the benefit versus the potential risk must be assessed prior to their use.[57,58]

Treatment. Most pregnant patients with kidney stones can be managed conservatively and will respond to intravenous isotonic crystalloids and analgesia.[55] However, kidney stones in pregnancy do carry an increased risk of premature delivery.[59] If urolithiasis is diagnosed or strongly suspected, consultation with a urologist and/or obstetrician (if viable pregnancy) is appropriate.

The febrile patient with urolithiasis should raise suspicion for concomitant pyelonephritis. The workup in these patients should also include blood and urine cultures and admission for intravenous antibiotics. Infected stones may need to be removed to cure the infection and avoid catastrophic complications such as gram-negative sepsis.[60]

When the Fetus is Viable. When abdominal pain is present in a woman with a viable fetus (usually considered more than 24 weeks gestation or an estimated fetal weight of 500 g or more), a detailed and accurate history is important. Vaginal bleeding is of particular concern in this population, as it may be indicative of placenta previa or abruptio placentae. Additionally, vaginal bleeding could represent the "bloody show" that often is present in labor. It is important to ascertain by history the presence or absence of leakage of fluid that may represent premature rupture of membranes. Ideally, all patients with a viable fetus should be evaluated while undergoing fetal monitoring, especially if the patient is reporting abdominal pain or vaginal bleeding. If such a patient is being evaluated in the ED, portable fetal monitoring equipment and the appropriate staff should be requested to come to the patient's bed and apply the monitoring devices. In addition to this, it is important to remember that caution should be exercised when performing a pelvic examination in the second half of pregnancy. In most instances, the emergency practitioner should not perform pelvic examinations in pregnant patients with a viable fetus.

Placenta previa should first be ruled out with abdominal ultrasound prior to performing a digital or speculum examination. If a digital exam is performed, sterile gloves should always be used to prevent infection, which may lead to an increased risk of premature rupture of membranes. If a speculum examination is performed, any fluid present in the vaginal vault should be tested for pH to rule out premature rupture of membranes, defined as the rupture of membranes prior to the onset of labor. Fluid in the vaginal vault with pH > 6.5 is indicative of amniotic fluid. Microscopic examination should also be performed to look for ferning, which is also indicative of amniotic fluid. If premature rupture of membranes is diagnosed, consultation with an obstetrician is appropriate to guide further management.[61]

Abruptio placentae deserves special mention due to its often

catastrophic outcomes, although isolated right lower quadrant pain would be unusual in this setting. Placental abruption is premature separation of the placenta from the uterine wall.[62] Risk factors for placental abruption include: maternal hypertension (the most common cause of abruption), maternal trauma, smoking, use of ethanol, use of cocaine, sudden decompression of the uterus, a short umbilical cord, post-amniocentesis, and advanced maternal age. Women with abruptio placentae typically present with vaginal bleeding, increased resting uterine tone, uterine tenderness, and hypertonic contractions. Fetal distress is common. Often, abruption is mistaken for the onset of labor, and delays in its diagnosis lead to increased maternal and fetal morbidity and mortality.

The diagnosis of placental abruption is a clinical one. Vaginal bleeding may or may not be present, depending on the size of the abruption.[62] A concealed abruption involves only partial placental separation and does not lead to vaginal bleeding. Abdominal ultrasound findings in abruption are highly variable and unreliable when no abruption is visualized.[34] Sensitivity and specificity have been reported as 24% and 96% based on a retrospective cohort study.[57] As many as 50% of placental abruptions may not be visualized on ultrasound, and this modality should never be used as a "rule out" study.[34,57] Patients with suspected placental abruption warrant immediate fetal monitoring and urgent obstetrical consultation and should not be sent outside of the emergency department for any diagnostic imaging.[34] Laboratory workup should include a CBC, coagulation panel, type and crossmatch, Rh status, and renal function studies.

Miscellaneous Causes of RLQ Pain in Females

Tables 4-6 include diagnoses that can result in right lower quadrant pain but are not exclusive to females. An in-depth discussion of each of these conditions is beyond the scope of this article, but some highlights are included.

Conclusion

Abdominal pain in females, especially in either lower quadrant, poses unique diagnostic challenges due to the presence of the reproductive organs and the possibility of a complication of pregnancy. A careful history, coupled with a thorough physical examination and the thoughtful use of the laboratory and imaging studies, will very often yield the correct diagnosis. As always, it is paramount to ensure that life-threatening diagnoses are considered and excluded first.

The accompanying algorithm (*see Figure 1*) should assist clinicians when approaching female patients with right lower quadrant abdominal pain.

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(continued on page 178)

Table 4. Additional Causes of RLQ Pain in Women of Child-Bearing Age

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Mittelschmerz	Cyclic pelvic pain	Normal laboratory and imaging studies	Prostaglandin synthetase inhibitors (non-steroidal antiinflammatories) have been used Can follow up with gynecologist
Dysmenorrhea	Painful menses - cramping pain Pain can radiate to anterior thigh or sacral regions	Normal studies	Prostaglandin synthetase inhibitors, oral contraceptive pills, and non-steroidal antiinflammatories have been used[63] Can follow up with gynecologist
Endometriosis	Dysmenorrhea, dyschezia, dyspareunia Chronic pelvic pain Examination may be normal	May have anemia if menometrorrhagia Laparoscopy is diagnostic	Agents that suppress ovulation have been used Can follow up with gynecologist
Uterine Mass/ Malignancy Fibroids	Pelvic pain and/or mass	May have anemia if menometrorrhagia Ultrasound CT scan	Refer to a gynecologist May need to consult gynecology in the ED if significant bleeding.
Cervical cancer	Abnormal uterine bleeding (postmenopausal, postcoital) May be found incidentally on pelvic examination	Ultrasound or CT scan to define mass	Arrange prompt outpatient workup and definitive diagnosis. May need to consult gynecology in the ED if significant bleeding.
Gastroenteritis (viral and bacterial)	May have sick contacts or be epidemic Exposure to raw/uncooked, or poorly refrigerated food Nausea, vomiting, and diarrhea	May show signs of dehydration Imaging usually normal	Oral or intravenous rehydration. Most are self-limited and do not require antibiotics Antimotility agents generally not needed nor recommended Consider admitting those in the extremes of age and the immunocompromised
Hernia	Palpable mass/bulge Signs/symptoms of obstruction if incarcerated Signs/symptoms of infection/peritonitis if strangulated	Acute abdominal series if obstruction suspected CT scan Ultrasound can help define the mass	Attempt reduction with sedation, analgesia, and Trendelenburg position Consult surgery if incarcerated or strangulated Refer to surgery if symptomatic (pain) but no complications
Musculoskeletal	Pain worse with certain positions or movements	Normal laboratory and imaging studies	Pain control: acetaminophen and NSAIDs, opioids Most are treated as outpatients. Consider admission if: neurologic deficit, elderly, poor social support, suspicion of infection or malignancy
Herpes Zoster	Burning pain Rash may be present (vesicles with erythematous base in crops); dermatomal distribution	Clinical diagnosis	Topical analgesics and antipruritics. Consider treating with antivirals (acyclovir, valacyclovir, famciclovir) the immunocompromised and those > 50 years old. Antivirals (decrease incidence) and vaccination (preventive) for post-herpetic neuralgia

(*Continued*)

Table 4. Additional Causes of RLQ Pain in Women of Child-Bearing Age (continued)

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Pyelonephritis	Flank pain, fever, shaking chills, nausea, vomiting, dysuria Costovertebral angle (CVA) tenderness	Urinalysis shows infection (WBCs, nitrites, leukocyte esterase) CT scan without IV contrast may show stones; with contrast may show stranding, perinephric abscess or gas (emphysematous pyelonephritis)	Antibiotics Supportive: rest, antipyretics, hydration, antiemetics, analgesics. Admit those who: look toxic, do not tolerate oral intake, failed outpatient treatment, are pregnant, have unstable vital signs, significant comorbidities, unsafe social situation.
Epiploic appendagitis	Can mimic acute appendicitis	Ultrasound can show noncompressible oval mass next to the colon CT scan with oral and IV contrast	Analgesia as needed Spontaneously resolves. Ensure that surgical diagnoses, especially acute appendicitis, are excluded.
Porphyria	Intermittent abdominal pain, neuropathies, constipation, personality changes	Elevated urine porphyrins (including porphobilinogen) Imaging is usually normal	Admit all except those with mild attacks High carbohydrate diet, intravenous glucose. Hematin 4 mg/kd/day Goal is to decrease heme synthesis and the production of porphyrin precursors Narcotics for pain.
Irritable bowel syndrome	Recurrent abdominal pain and bloating Change in stool frequency and/or consistency Diagnosis follows the Rome criteria (Recurrent abdominal pain or discomfort 3 days per month in the last 3 months associated with 2 or more of the following: improvement with defecation; and/or onset associated with a change in stool frequency; and/or onset associated with a change in form [appearance] of stool.)	Normal laboratory and imaging studies	Often need outpatient multifaceted approach: antispasmodics for cramping, antidepressants, loperamide for diarrhea, patient support groups and other non-pharmacologic interventions may help.[63]
Inflammatory bowel disease	Bloody diarrhea and abdominal pain Signs and symptoms of obstruction Weight loss Fatigue Crohn's: fistulas	CT scan with oral and IV contrast: inflammation, obstruction, megacolon (ulcerative colitis) Anemia Extraintestinal manifestations: arthritides, rashes	Admit if : dehydrated, uncontrolled pain, unable to tolerate food, failure of outpatient treatment, strictures with obstruction, abscess. Refer for colonoscopy if no complications Aminosalicylic acid derivatives are first choice, steroids next. Immunomodulators have also been used.
Psychogenic	Normal or inconsistent physical examination	Normal laboratory and imaging studies	Exclude all life-threatening, disabling, and treatable medical conditions Provide emotional support to patient and family Refer to a primary care physician and to a psychiatrist

Table 5. Additional Causes of RLQ Pain in Pre-Menarchal Females

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Meckel's diverticulum	Abdominal pain and lower gastrointestinal bleeding (can be painless) Signs of intestinal obstruction Signs of diverticular inflammation	Anemia if significant bleeding Plain films may show obstruction Meckel scan (technetium Tc99m pertechnetate scintiscan) will detect gastric tissue in the Meckel's	Supportive treatment. May need transfusions if significant bleeding Consult surgery for admission and resection
Intussusception	Colicky abdominal pain and vomiting Asymptomatic periods Lethargy (late) "Currant jelly" stools Can have sausage-shaped mass in the right upper quadrant	Contrast enema (can be diagnostic and therapeutic) Ultrasound can visualize intussusceptum Plain films can show obstruction or perforation	Supportive treatment. Evaluate for a mechanical lead point (mass). Admit even if reduction is achieved with barium enema, as up to 10% can have recurrence in the next 24 hours. Reduction usually made in consultation with a surgeon, as complications may arise that may require immediate surgery.
Henoch Schonlein purpura	Abdominal pain (colicky), bloody diarrhea, arthralgias, rash (palpable purpura, more in buttocksand legs), nephritis (hematuria and/or proteinuria)	Urinalysis with hematuria and/or proteinuria CBC with leukocytosis and thrombocytosis Serum IgA levels are increased in 50% of patients Plain films and CT scan can show obstruction or intussusception	Supportive management Can use NSAIDs for pain control. Admit those who appear ill, have severe pain, significant gastrointestinal bleeding, and those with renal involvement; some require steroids.
Cyclic vomiting/ abdominal migraine (migraine equivalent)	Family history of migraine is common Paroxysmal mid-abdominal pain and vomiting. Aura and headaches can be minimal or absent.	Normal laboratory and imaging studies	Antiemetics may help with the acute attack. Most resolve with sleep. Refer to a neurologist to establish the diagnosis, as some may require prophylaxis
Urinary tract infection	Urgency, frequency, hesitancy Dysuria Hematuria No fever	Urinalysis with white blood cells, nitrites, leukocyte esterase Positive urine culture	Most can be treated as outpatients with 3 days of antibiotics: Bactrim is first choice when local resistance is low; fluoroquinolones if resistance is high or infection is complicated; nitrofurantoin (Macrodantin); amoxicillin/clavulanate (Augmentin) Can offer a urinary analgesic if dysuria is significant, like (phenazopyridine [Pyridium]) Admit the elderly or immunocompromised who may present with a sepsis syndrome. Treat pregnant women for up to 14 days. Cephalexin (Keflex) is commonly used.
Mesenteric adenitis	Acute right lower quadrant pain May be indistinguishable from acute appendicitis Fever	Ultrasound with graded compression: normal appendix and enlarged lymph nodes CT scan with oral and IV contrast Is a diagnosis of exclusion	Self-limited disease Make sure surgical causes of abdominal pain are excluded, especially acute appendicitis.

(*Continued.*)

Table 5. Additional Causes of RLQ Pain in Pre-Menarchal Females (Continued)

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Toxic, i.e., lead poisoning	Changes in behavior Abdominal pain with constipation	Anemia Elevated free erythrocyte protoporphyrin levels Elevated blood lead level Radiographs may show lead lines (children) or radio-opaque foreign bodies in the gastrointestinal tract	Decontaminate as needed Admit to ICU those with encephalopathy Chelation indicated for children with levels between 45-70 mcg/dL and adults between 70-100 mcg/dL Chelators: BAL, calcium disodium EDTA succimer Remove from the source

Table 6. Additional Causes of RLQ Pain in Post-Menopausal Females

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Abdominal aortic aneurysm (AAA)	Most are asymptomatic until rupture Can present with back, flank, or groin pain Can present with syncope or shock A pulsatile abdominal mass is virtually diagnostic, but not often found	Ultrasound is sensitive, good for screening, can be performed at the bedside CT scan is best for details but requires a stable patient	Treat shock if present Type and crossmatch Control blood pressure and heart rate to decrease vessel wall tension Immediate vascular surgery consultation for all symptomatic AAAs
Diverticulosis/itis (typically LLQ)	History of constipation or recent change in bowel movements Fever if infected May have lower gastrointestinal bleeding with R sided diverticuli	Leukocytosis if infected Anemia if significant bleeding Acute abdominal series may show free air if perforated CT scan with IV and oral contrast	Those with mild diverticulitis: can be discharged with liquid diet and oral antibiotics (ciprofloxacin and metronidazole are commonly used) Admit if: toxic appearing, fails out-patient management, poor pain control, peritonitis, immunocompromised, unable to tolerate food. Diverticular abscesses > 5 cm need to be drained
Intestinal ischemia	Severe abdominal pain with paucity of clinical findings Can have symptoms of "intestinal angina" Nausea, vomiting, and diarrhea are commonly present Advanced ischemia/necrosis: distention, ileus, peritonitis, shock Risk factors: atrial fibrillation, hypoperfusion, vasoconstriction, atherosclerosis, hypercoagulable states	Laboratory studies are often nonspecific and unreliable until late in the course of the disease -leukocytosis -elevated lactate -elevated D-dimer Plain films are nonspecific or normal -may show perforation or obstruction -pneumatosis intestinalis, portal vein gas, and thumbprinting are late Multidetector CT scans are good at detecting mesenteric ischemia Angiography is the gold standard, and can be both diagnostic and therapeutic	Intestinal angina: no effective treatment exists, but angioplasty with/without stenting has been used, as well as mesenteric revascularization Angiography can be both diagnostic and therapeutic in cases of thrombosis or embolism Non-occlusive ischemia: identify and correct the underlying cause (vasospasm, hypovolemia, low flow state) Laparotomy is indicated to remove necrotic bowel. Admit all patients to the Intensive Care Unit.

(*Continued*)

Table 6. Additional Causes of RLQ Pain in Post-Menopausal Females (continued)

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
GI malignancy - colon cancer	Abdominal pain, altered bowel habits, change in stool caliber, occult blood, bowel signs and symptoms of obstruction	CT scan will show mass, obstruction, and presence of metastases	Arrange prompt outpatient workup and definitive diagnosis. May need admission if obstructed or significant bleeding
Obstruction small bowel	Vomiting, abdominal pain and distention Absence of bowel movements and/or flatus	Plain films (acute abdominal series): dilated bowel loops with air-fluid levels; absent colonic gas; free air if perforated CT scan: dilated bowel loops adjacent to collapsed loops	Place nasogastric tube for decompression. Volume resuscitate Use antiemetics and analgesics as needed. Early surgical consultation for admission and further management
Obstruction Small bowel Large bowel Volvulus Sigmoid Cecal Intussusception Acute colonic pseudo-obstruction (Ogilvie's syndrome)	Vomiting, abdominal pain and distention Absence of bowel movements and/or flatus Sudden onset of symptoms may represent a volvulus	Plain films (acute abdominal series): dilated colon; "kidney bean" appearance in volvulus; free air if perforated CT scan	Supportive management: fluid resuscitation, nasogastric tube placement, use of analgesia and antiemetics Antibiotics are generally recommended Early surgical consultation and admission Sigmoidoscopy can reduce a sigmoid volvulus Pesudo-obstruction is managed with bowel rest, intravenous hydration, and treating the underlying disorders.

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Physician CME Questions

11. Risk factors for PID include the following *except*:
    A. age < 25 years.
    B. age of first intercourse < 15 years.
    C. lower socioeconomic status.
    D. being married.
    E. multiple sexual partners.

12. When suspecting TOA as the most likely diagnosis, the best initial imaging modality is the ultrasound.
    A. True
    B. False

13. All of the following statements about adnexal torsion are true *except*:
    A. Ovarian masses and tumors increase the likelihood of torsion.
    B. Adnexal torsion is a gynecological surgical emergency.
    C. Women undergoing assisted reproductive therapy have a higher than normal risk for torsion.
    D. Most torsions occur after the menopause.

14. A normal Doppler flow reliably excludes the diagnosis of adnexal torsion.
    A. True
    B. False

15. Risk factors for ectopic pregnancy include the following *except*:
    A. multiple previous pregnancies.
    B. previous PID.
    C. previous or current IUD use.
    D. previous tubal surgeries.

16. The classic triad in ectopic pregnancy consists of:
    A. amenorrhea, vomiting, and fever.
    B. abdominal pain, fever, and breast tenderness.
    C. amenorrhea, abdominal pain, and abnormal vaginal bleeding.
    D. vaginal bleeding, irregular menses, and dyspareunia.

17. Which of the following is the correct definition of threatened miscarriage?
    A. Vaginal bleeding, a closed cervical os, and positive heartbeat on ultrasound
    B. Passage of tissues with a closed cervical os before week 20
    C. Passage of products of conception, a closed cervical os, and visible retained tissue on ultrasound
    D. Vaginal bleeding with an open cervix but no passage of tissue

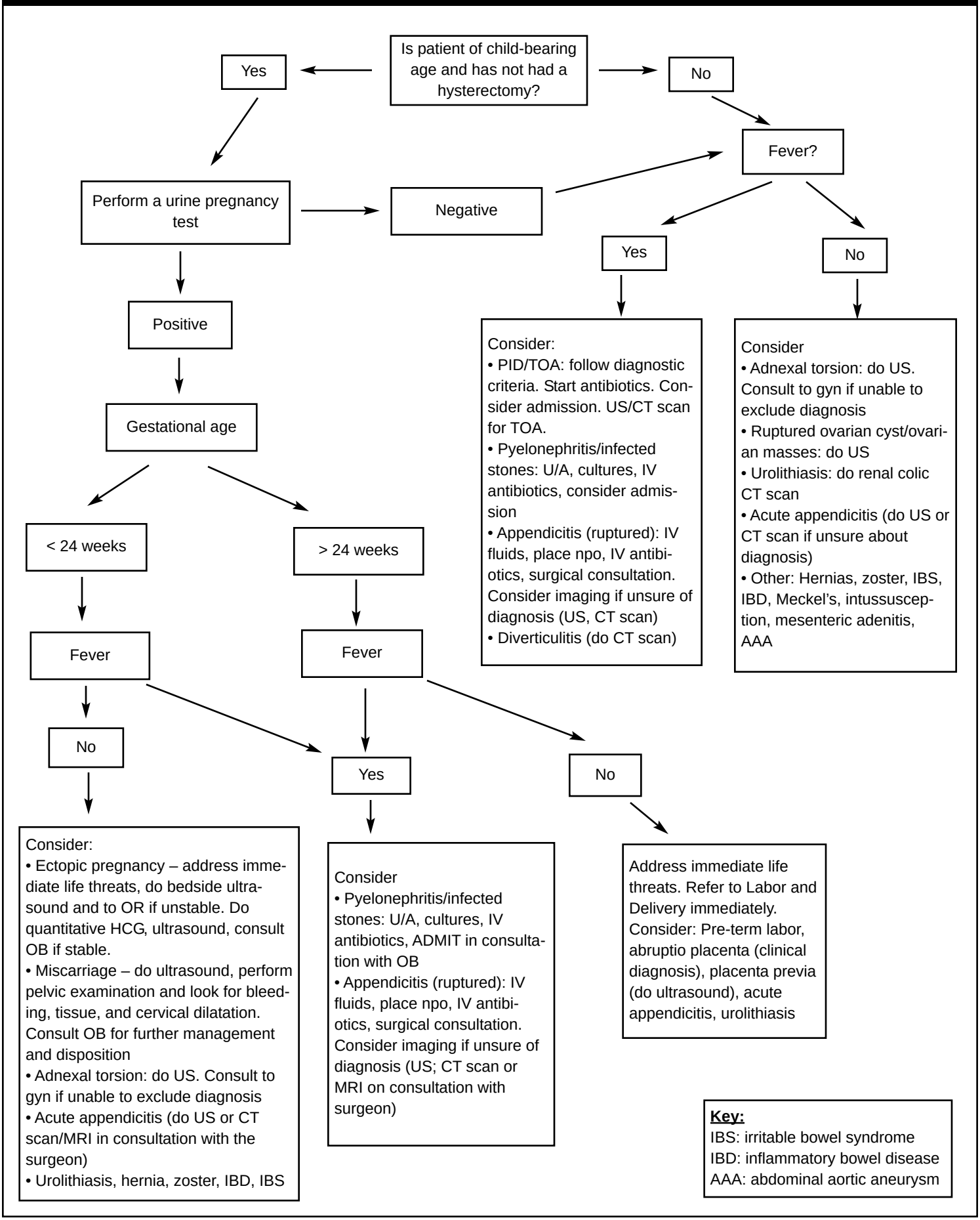
18. The guidelines on diagnostic imaging in pregnancy set forth by the American College of Obstetrics and Gynecology include which of the following?:
    A. Exposures < 5 rads have not been associated with increased fetal anomalies or spontaneous abortion.
    B. Ultrasound and MRI have not been associated with any fetal adverse effects.
    C. Therapeutic radioactive iodine isotopes are contraindicated.
    D. Maternal health should not be compromised by fears of the dangers of radiation.
    E. All of the above are true.

19. Which of the following statements is true regarding placental abruptio?
    A. Fetal distress is not common.
    B. Vaginal bleeding is pathognomonic of an abruption.
    C. The diagnosis is a clinical one.
    D. It is very difficult to confuse placental abruption with the onset of labor, as the presentation is different.

20. Which of the following statements regarding appendicitis in pregnancy is *not true*?
    A. Abdominal ultrasound is a useful definitive imaging modality when appendicitis is highly suspected.
    B. The incidence of appendicitis is increased in pregnant patients as compared to the general population.
    C. The white blood cell count is of limited utility in diagnosing acute appendicitis.
    D. Fetal and maternal morbidity and mortality increase substantially once appendiceal rupture occurs.

CME Answer Key: 11. D; 12. A; 13. D; 14. B; 15. A; 16. C; 17. A; 18. E; 19. C; 20. A

Figure 1. Approach to the Patient with RLQ Pain



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RLQ Abdominal Pain in Females

Additional Causes of RLQ Pain in Women of Child-Bearing Age

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Mittelschmerz	Cyclic pelvic pain	Normal laboratory and imaging studies	Prostaglandin synthetase inhibitors (non-steroidal antiinflammatories) have been used Can follow up with gynecologist
Dysmenorrhea	Painful menses - cramping pain Pain can radiate to anterior thigh or sacral regions	Normal studies	Prostaglandin synthetase inhibitors, oral contraceptive pills, and non-steroidal antiinflammatories have been used Can follow up with gynecologist
Endometriosis	Dysmenorrhea, dyschezia, dyspareunia Chronic pelvic pain Examination may be normal	May have anemia if menometrorrhagia Laparoscopy is diagnostic	Agents that suppress ovulation have been used Can follow up with gynecologist
Uterine Mass/ Malignancy Fibroids	Pelvic pain and/or mass	May have anemia if menometrorrhagia Ultrasound CT scan	Refer to a gynecologist May need to consult gynecology in the ED if significant bleeding.
Cervical cancer	Abnormal uterine bleeding (postmenopausal, postcoital) May be found incidentally on pelvic examination	Ultrasound or CT scan to define mass	Arrange prompt outpatient workup and definitive diagnosis. May need to consult gynecology in the ED if significant bleeding.
Gastroenteritis (viral and bacterial)	May have sick contacts or be epidemic Exposure to raw/uncooked, or poorly refrigerated food Nausea, vomiting, and diarrhea	May show signs of dehydration Imaging usually normal	Oral or intravenous rehydration. Most are self-limited and do not require antibiotics Antimotility agents generally not needed nor recommended Consider admitting those in the extremes of age and the immunocompromised
Hernia	Palpable mass/bulge Signs/symptoms of obstruction if incarcerated Signs/symptoms of infection/peritonitis if strangulated	Acute abdominal series if obstruction suspected CT scan Ultrasound can help define the mass	Attempt reduction with sedation, analgesia, and Trendelenburg position Consult surgery if incarcerated or strangulated Refer to surgery if symptomatic (pain) but no complications
Musculoskeletal	Pain worse with certain positions or movements	Normal laboratory and imaging studies	Pain control: acetaminophen and NSAIDs, opioids Most are treated as outpatients. Consider admission if: neurologic deficit, elderly, poor social support, suspicion of infection or malignancy
Herpes Zoster	Burning pain Rash may be present (vesicles with erythematous base in crops); dermatomal distribution	Clinical diagnosis	Topical analgesics and antipruritics. Consider treating with antivirals (acyclovir, valacyclovir, famciclovir) the immunocompromised and those > 50 years old. Antivirals (decrease incidence) and vaccination (preventive) for post-herpetic neuralgia

(*Continued*)

Additional Causes of RLQ Abdominal Pain in Women of Child-Bearing Age (continued)

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Pyelonephritis	Flank pain, fever, shaking chills, nausea, vomiting, dysuria Costovertebral angle (CVA) tenderness	Urinalysis shows infection (WBCs, nitrites, leukocyte esterase) CT scan without IV contrast may show stones; with contrast may show stranding, perinephric abscess or gas (emphysematous pyelonephritis)	Antibiotics Supportive: rest, antipyretics, hydration, antiemetics, analgesics. Admit those who: look toxic, do not tolerate oral intake, failed outpatient treatment, are pregnant, have unstable vital signs, significant comorbidities, unsafe social situation.
Epiploic appendagitis	Can mimic acute appendicitis	Ultrasound can show noncompressible oval mass next to the colon CT scan with oral and IV contrast	Analgesia as needed Spontaneously resolves. Ensure that surgical diagnoses, especially acute appendicitis, are excluded.
Porphyria	Intermittent abdominal pain, neuropathies, constipation, personality changes	Elevated urine porphyrins (including porphobilinogen) Imaging is usually normal	Admit all except those with mild attacks High carbohydrate diet, intravenous glucose. Hematin 4 mg/kd/day Goal is to decrease heme synthesis and the production of porphyrin precursors Narcotics for pain.
Irritable bowel syndrome	Recurrent abdominal pain and bloating Change in stool frequency and/ or consistency Diagnosis follows the Rome criteria (Recurrent abdominal pain or discomfort 3 days per month in the last 3 months associated with 2 or more of the following: improvement with defecation; and/or onset associated with a change in stool frequency; and/or onset associated with a change in form [appearance] of stool.)	Normal laboratory and imaging studies	Often need outpatient multifaceted approach: antispasmodics for cramping, antidepressants, loperamide for diarrhea, patient support groups and other non-pharmacologic interventions may help.
Inflammatory bowel disease	Bloody diarrhea and abdominal pain Signs and symptoms of obstruction Weight loss Fatigue Crohn's: fistulas	CT scan with oral and IV contrast: inflammation, obstruction, megacolon (ulcerative colitis) Anemia Extraintestinal manifestations: arthritides, rashes	Admit if : dehydrated, uncontrolled pain, unable to tolerate food, failure of outpatient treatment, strictures with obstruction, abscess. Refer for colonoscopy if no complications Aminosalicylic acid derivatives are first choice, steroids next. Immunomodulators have also been used.
Psychogenic	Normal or inconsistent physical examination	Normal laboratory and imaging studies	Exclude all life-threatening, disabling, and treatable medical conditions Provide emotional support to patient and family Refer to a primary care physician and to a psychiatrist

Additional Causes of RLQ Pain in Pre-Menarchal Females

CONDITION	HISTORY/PHYSICAL EXAMINATION	LABORATORIES/IMAGING	MANAGEMENT/DISPOSITION
Meckel's diverticulum	Abdominal pain and lower gastrointestinal bleeding (can be painless) Signs of intestinal obstruction Signs of diverticular inflammation	Anemia if significant bleeding Plain films may show obstruction Meckel scan (technetium Tc99m pertechnetate scintiscan) will detect gastric tissue in the Meckel's	Supportive treatment. May need transfusions if significant bleeding Consult surgery for admission and resection
Intussusception	Colicky abdominal pain and vomiting Asymptomatic periods Lethargy (late) "Currant jelly" stools Can have sausage-shaped mass in the right upper quadrant	Contrast enema (can be diagnostic and therapeutic) Ultrasound can visualize intussusceptum Plain films can show obstruction or perforation	Supportive treatment. Evaluate for a mechanical lead point (mass). Admit even if reduction is achieved with barium enema, as up to 10% can have recurrence in the next 24 hours. Reduction usually made in consultation with a surgeon, as complications may arise that may require immediate surgery.
Henoch Schonlein purpura	Abdominal pain (colicky), bloody diarrhea, arthralgias, rash (palpable purpura, more in buttocksand legs), nephritis (hematuria and/or proteinuria)	Urinalysis with hematuria and/or proteinuria CBC with leukocytosis and thrombocytosis Serum IgA levels are increased in 50% of patients Plain films and CT scan can show obstruction or intussusception	Supportive management Can use NSAIDs for pain control. Admit those who appear ill, have severe pain, significant gastrointestinal bleeding, and those with renal involvement; some require steroids.
Cyclic vomiting/ abdominal migraine (migraine equivalent)	Family history of migraine is common Paroxysmal mid-abdominal pain and vomiting. Aura and headaches can be minimal or absent.	Normal laboratory and imaging studies	Antiemetics may help with the acute attack. Most resolve with sleep. Refer to a neurologist to establish the diagnosis, as some may require prophylaxis
Urinary tract infection	Urgency, frequency, hesitancy Dysuria Hematuria No fever	Urinalysis with white blood cells, nitrites, leukocyte esterase Positive urine culture	Most can be treated as outpatients with 3 days of antibiotics: Bactrim is first choice when local resistance is low; fluoroquinolones if resistance is high or infection is complicated; nitrofurantoin (Macrodantin); amoxicillin/clavulanate (Augmentin) Can offer a urinary analgesic if dysuria is significant, like (phenazopyridine [Pyridium]) Admit the elderly or immunocompromised who may present with a sepsis syndrome. Treat pregnant women for up to 14 days. Cephalexin (Keflex) is commonly used.
Mesenteric adenitis	Acute right lower quadrant pain May be indistinguishable from acute appendicitis Fever	Ultrasound with graded compression: normal appendix and enlarged lymph nodes CT scan with oral and IV contrast Is a diagnosis of exclusion	Self-limited disease Make sure surgical causes of abdominal pain are excluded, especially acute appendicitis.
Toxic, i.e., lead poisoning	Changes in behavior Abdominal pain with constipation	Anemia Elevated free erythrocyte protoporphyrin levels Elevated blood lead level Radiographs may show lead lines (children) or radio-opaque foreign bodies in the gastrointestinal tract	Decontaminate as needed Admit to ICU those with encephalopathy Chelation indicated for children with levels between 45-70 mcg/dL and adults between 70-100 mcg/dL Chelators: BAL, calcium disodium EDTA succimer Remove from the source

Pelvic Inflammatory Disease: CDC Criteria 2006

MINIMUM CRITERIA	ADDITIONAL CRITERIA
• Abdominal pain • Cervical motion tenderness • Adnexal tenderness	• Fever > 101°F (38.3°C) • Abnormal cervical/vaginal mucopurulent discharge • Elevated erythrocyte sedimentation rate and/or C-reactive protein • Presence of abundant numbers of white blood cells on saline microscopy of vaginal secretions • Positive cervical cultures with *N. gonorrhoeae* or *C. trachomatis*

Intravenous Antibiotic Recommendations for PID, CDC

IV Regimen A	Cefotetan (Cefotan) 2 g IV every 12 hours **Or** Cefoxitin (Mefoxin) 2 g IV every 6 hours **Plus** Doxycycline 100 mg orally (preferred) or IV every 12 hours
IV Regimen B	Clindamycin (Cleocin) 900 mg IV every 8 hours **Plus** Gentamycin loading dose IV or IM (2 mg/kg of body weight), followed by maintenance dose (1.5 mg/kg) every 8 hours
Alternative IV Regimen (pen/ceph allergic)	Ampicillin/sulbactam (Unasyn) 3 g IV every 6 hours **Plus** Doxycycline 100 mg orally (preferred) or IV every 12 hours

Outpatient Antibiotic Recommendations for PID, CDC

Ceftriaxone 250 mg IM as single dose
PLUS
Doxycycline 100 mg orally twice daily for 14 days (with or without metronidazole [Flagyl] 500 mg orally every 12 hours for 14 days)
OR
Cefoxitin 2 g IM in a single dose
AND
Probenecid 1 g orally administered concurrently in a single dose
PLUS
Doxycycline 100 mg orally every 12 hours for 14 days (with or without metronidazole [Flagyl] 500 mg orally every 12 hours)

Supplement to *Emergency Medicine Reports*, July 9, 2007: "Right Lower Quadrant Pain in Females." *Authors:* **Larissa I. Velez, MD, FACEP**, Associate Program Director, Emergency Medicine, University of Texas Southwestern, Staff Toxicologist, North Texas Poison Center, Dallas; **Fernando Benitez, MD, FACEP**, Department of Emergency Medicine, University of Texas Southwestern, Dallas; **J. Clinton Lowry, MD**, University of Texas Southwestern, Dallas; and **Rolando Diaz, MD**, University of Texas Southwestern, Dallas.
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Traumatic Cardiopulmonary Arrest

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Trauma is the leading cause of death in patients between the ages of 1 and 44 years and is the fifth leading cause of overall deaths in the United States. The annual cost to care for trauma is estimated at between $260 and $440 billion. Each year, more than 150,000 patients die from traumatic injuries. It is estimated that between one-third[1] and one-half[2] of these deaths occur in the out-of-hospital environment and that up to 80% of these deaths occur within the first few hours of injury.[3] This suggests that many emergency medical technicians, emergency physicians, and trauma surgeons will face the challenge of traumatic cardiopulmonary arrest following traumatic injury as a part of their clinical practice. This article reviews the incidence, cause, and factors that impact survival following traumatic cardiopulmonary arrest.

— The Editor

Introduction

The decision to resuscitate a patient with traumatic cardiopulmonary arrest is not as simple as it may first appear. The course of action is only clear when the patient demonstrates signs of prolonged loss of vital functions (rigor mortis, dependent lividity) or an obvious unsurvivable injury (decapitation, hemicorporectomy). Many of these patients are young and in their most productive years of life; this creates an argument for aggressive management. This not only charges our decisions with an emotional element, but also implies that even limited success in the number of lives saved may reap large benefits in terms of potential economic benefit.

On the other hand, survival from traumatic cardiopulmonary arrest is unusual,[4-7] and the costs of futile resuscitation efforts can be significant. In addition, the healthcare provider must be cognizant of other concerns such as risk of exposure to blood and body fluids, consumption of valuable resources such as blood products, and risk of injury to pre-hospital providers during rapid transport.

The purpose of this article is to review the incidence and caus-

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es of traumatic cardiopulmonary arrest, identify factors that impact the outcome of these patients, and suggest a rationale approach to the management of traumatic cardiopulmonary arrest both in and out of the hospital environment.

Incidence of Trauma Arrest

The exact incidence of traumatic cardiopulmonary arrest is difficult to determine. There are an estimated 60 million people injured annually in the United States; approximately 5-10% of these patients are injured seriously. This equates to 3-6 million patients with serious injury yearly. This number can be validated by a derivation that suggests one in six of the 29.5 million patients admitted to the hospital for trauma (4.9 million hospitalizations) will have a serious injury.[8] If we assume that 80% of the 150,000 trauma deaths occur in either the out-of-hospital setting or during early resuscitation,[1-3] we can estimate that 2-4 patients out of 100 with significant trauma will suffer a traumatic cardiopulmonary arrest.

This derived estimate seems to track well with two large studies. Battistella and coworkers[9] described 604 traumatic cardiopulmonary arrests among 16,724 patients admitted to their Level I trauma center. This suggests 3.6 trauma arrests for every 100 admissions. Similarly, Stockinger and McSwain[10] found 588 traumatic cardiopulmonary arrests among 16,651 Level I trauma admissions or 3.5 trauma arrests per 100 admissions.

It is of interest that Falcone and colleagues[11] found 320 traumatic cardiopulmonary arrests among 12,518 air medical transports, or 2.6 trauma arrests per 100 transports. Presumably, many of the transported patients were not exclusively trauma patients. Margolin et al[12] found only 67 traumatic cardiopulmonary arrests among 14,905 transports. This represents a significantly lower incidence of 4.5 trauma arrests per 1000 transports and also may reflect a difference in referral patterns for air transport in their region.

Gathering more specific estimates in the scientific literature is difficult for several reasons. First, the definition of traumatic cardiopulmonary arrest varies among studies with regard to the inclusion of non-trauma diagnosis such as asphyxia, drowning, hanging, electrocution, and major burns. Many other studies include only those patients taken to trauma centers. Thus, they may underestimate the true incidence of traumatic cardiopulmonary arrest by excluding patients with early out-of-hospital death who may be pronounced in the field or transported to non-trauma hospitals. Other studies fail to identify the total number of patients admitted to the study facility or catchment area,[7,13,14] thus making any population or admission-based estimates difficult. Finally, many other authors tend to focus on segmented populations or specific mechanisms of injury,[15] which makes a true determination of incidence impossible.

Epidemiology of Trauma Arrest

It is important to understand the exact causes of early trauma deaths so that one can implement treatment protocols that address the most common causes of traumatic cardiopulmonary arrest. Baker and coworkers[16] were the first to describe the pattern of early trauma deaths. Blunt injuries caused 53% of deaths, and penetrating trauma caused an additional 40%. More than one-half of the patients (53%) died prior to arrival to the hospital, and 29% died in the first 48 hours. Sauaia et al[1] conducted a similar study in Denver. They found that 49% of deaths were due to penetrating trauma and 48% were due to blunt injury. In their study, 34% of deaths occurred before reaching the hospital and 53% died within the first 48 hours of admission. Patients with penetrating injury who reached the hospital tended to die early (first 48 hours), whereas those with blunt trauma typically suffered late deaths. Almost half (52%) of prehospital deaths were due to central nervous system (CNS) injury, and an additional 36% were due to exsanguinations or a combination of both injury patterns. In patients who died in the first 48 hours of admission, exsanguination was the major cause of death. Trunkey[2] noted that most of the out-of-hospital deaths were caused by CNS (brain and spinal cord injury) and major vascular trauma whereas early deaths were due primarily to exsanguination. These findings were recently validated in a rural setting[17] in which 42% of deaths were related to CNS injury, 20% were due to exsanguinations, and 8% were due to airway compromise.

Correction

In the May/June 2007 issue of *Trauma Reports*, a sentence was noted incorrectly on page 3. It should have read: "Given the aging population of the United States, the incidence of hip fracture is estimated to exceed more than 500,000 cases by the year 2040."

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Table 1. Leading Causes of Trauma Deaths

PREHOSPITAL	EARLY HOSPITAL
Major head injury	Hemoperitoneum
Spinal cord injury	Massive hemothorax
Traumatic aortic rupture	Retroperitoneal hemorrhage from major pelvic fractures
Major vascular injury	
Cardiac rupture	
Airway compromise	

The major prehospital and early hospital causes of death are listed in Table 1.

Factors Affecting Survival

Overall, survival from traumatic cardiopulmonary arrest is low. The survival in those studies focusing on prehospital traumatic cardiopulmonary arrest varies from 0-2.9%. *(See Table 2.)* Although much of this information is derived from studies on emergency thoracotomy, several authors have examined overall survival for those patients in which resuscitation and transport is provided. Two additional points need to be made about studies of traumatic cardiopulmonary arrest. First, there is no standardized definition or terminology that exists for this field that mirrors the the Utstein criteria used in studies involving primary cardiac arrest.[18] At a minimum, a gold standard for reporting results should be the number or percent of neurologically intact survivors. Additionally, it must be remembered that the overall survival rate for primary cardiac arrest, when measured in terms of neurologically intact survivors, is not significantly better than traumatic cardiac arrest, particularly in urban environments.[19,20]

Shimazu and coworkers[4] examined survival among 267 victims of traumatic cardiopulmonary arrest (including 217 blunt injuries and 55 penetrating injuries). There were 7 (2.6%) survivors; 4 (1.5%) were neurologically intact. Stratton et al[21] examined 1051 victims of traumatic cardiopulmonary arrest, including 497 penetrating injuries and 382 blunt trauma victims. One hundred sixteen patients were excluded for various reasons, including loss of pulse in the field. The survival rate was 0.8% for penetrating injury and 1.6% for blunt injury. Battistella and colleagues[9] found an overall survival rate of 2.6%, with nearly one-half of the survivors having neurologic sequelae. Other studies by Fulton et al[22] (2.6%) and Pasquale et al[14] (2.9%) confirm these results.

Recent studies, however, have demonstrated more promising results. Willis and coworkers[23] reported a survival rate of 4.5% among trauma arrests transported to the hospital over a four-year period. Their methodology did not include victims pronounced on-scene and also included other non-traumatic diagnoses such as electrocution. Cera and coworkers[7] noted 15 surviving patients among 195 patients (7.7%) arriving at their trauma center with CPR in progress, including 34 patients in which no resuscitation was attempted. Lockey et al[24] found a survival of 7.5% among 909 trauma arrests transported by air medical transport. This included a significant number of patients who were pronounced dead in the field. Finally, Pickens et al[13] also found an overall survival rate of 7.7% among 184 patients transported to a Level I trauma center with CPR in progress. Overall, 3590 patients with traumatic cardiopulmonary arrest were reported in these studies, with 180 (5.1%) survivors.

Several authors have evaluated more specific criteria that can be used to identify those victims of traumatic cardiopulmonary arrest, criteria that can be used to predict survival or reduce futile resuscitative efforts. These are described below.

Initial Cardiac Rhythm

At least five studies have addressed the impact of underlying cardiac rhythm on survival. Esposito and colleagues[25] were able to document an underlying rhythm in 102 of their study population. There were two survivors in this group: one with an underlying ventricular dysrhythmia and one with sinus-based PEA (pulseless electrical activity). No patient with asystole survived. Fulton et al[22] found that patients with an underlying ventricular dysrhythmia or PEA had a better outcome than those with asystole or an idioventricular rhythm. In fact, no asystolic patients survived. Interestingly, however, no patient who was defibrillated upon arrival to the trauma center survived. This contrasts with a study by Aprahamian et al[26] in which only patients who required defibrillation in the emergency department survived. In their series of 604 trauma arrests, Battistella et al[9] found no survivors among

Table 2. Survival from Traumatic Cardiopulmonary Arrest

AUTHOR	MECHANISM	NO. PATIENTS	NO. SURVIVAL	COMMENT
Shimazu et al[4]	Blunt, penetrating	267	7 (2.6%)	Only included those patients transported to hospital; only 4 neurologically intact
Stratton et al[21]	Blunt, penetrating	879	9 (1.0%)	116 patients pronounced in field excluded; 3 neurologically intact
Battistella et al[9]	Blunt, penetrating	604	16 (2.7%)	All patients included; 7 neurologically impaired
Fulton et al[22]	Blunt, penetrating	245	6 (2.4%)	Only included those patients transported to hospital
Pasqualeet al[14]	Blunt, penetrating	106	3 (2.8%)	Only included those patients transported to hospital
Willis et al[23]	Blunt, penetrating	89	4 (4.5%)	Only included those patients transported to hospital
Cera et al[7]	Blunt, penetrating	195	53 (27.2%)	Only included those patients transported to hospital
Lockey et al[24]	Blunt, penetrating	909	68 (7.5%)	Only included air transported patients
Pickens et al[13]	Blunt, penetrating	266	14 (5.3%)	Only included patients transported to hospital

212 patients who had asystole and no long-term survivors among patients with PEA and a heart rate less than 40, although 5 of 134 were admitted. All 16 survivors came from the group including those with a heart rate of more than 40 beats per minute. Stratton and colleagues[21] also found that all neurologically intact survivors had PEA with an underlying rhythm of sinus tachycardia or normal sinus rhythm. On the other hand, Cera and coworkers[7] found that PEA with an underlying sinus rhythm was predictive of better survival but 11 survivors had PEA (20.7% of survivors) and 3 survivors had ventricular dysrhythmias (5.7%). Finally, Pickens and colleagues[13] found that an underlying rate of 40 or greater showed improved survival, although 43% of survivors had a rate of less than 40. Also, none of the patients who required defibrillation in the emergency department survived. This final point may reflect that patients with multiple traumatic cardiac arrests have a uniformly poor prognosis.[23]

Duration of CPR

There is significant evidence to suggest that the duration of chest compressions may have an impact on the outcome of traumatic cardiopulmonary arrest. Mattox et al[27] reviewed 100 consecutive cases of trauma (37 blunt and 63 penetrating) in patients who received more than 3 minutes of CPR. There were no survivors in these patients. One intriguing finding was the presence of fatal air embolism in the coronary arteries on autopsy in 12 patients with lung parenchymal injury. Durham and colleagues[28] noted a statistical difference in CPR time for survivors of stab wounds, gunshot wounds, and blunt trauma. They proposed a limit of 5 minutes for patient viability following trauma cardiopulmonary arrest. Pasquale et al[14] supported these findings in 89 patients who underwent more than 5 minutes of CPR following blunt or non-thoracic penetrating trauma. There were no survivors in this population. The two survivors in the Pasquale and coworker's study[14] both had CPR of less than 5 minutes and survived neurologically intact. Fulton et al[22] found no survivors among those patients who had CPR performed for longer than ten minutes or who sustained multiple cardiac arrests. However, they concluded that CPR should not be performed for longer than 30 minutes without response. This agrees with the findings of Falcone et al;[11] in their study of air transported patients, the average time of CPR was 33 minutes. There were no survivors in their study. On the other hand, more recent work by Pickens and colleagues[13] demonstrated that 3 of their 14 survivors had CPR in progress for more than 15 minutes. This last study suggests that although shorter duration of CPR is associated with better survival, prolonged CPR does not preclude a successful outcome. Fialka and coworkers[29] showed that the average duration of closed chest CPR was 13 minutes (range, 11-15 minutes) among their surviving patients.

Advanced Life Support (ALS) Measures

The role of prehospital ALS, including airway management and intravenous access, has been hotly debated in the trauma literature. The role of specific interventions, particularly endotracheal intubation, has been considered with regard to outcome from traumatic cardiopulmonary arrest. Copass et al[30] reported on 131 patients who required CPR following traumatic injury. They found that survivors were more likely to have successful endotracheal intubation (97% versus 65%) and intravenous access (100% versus 70%). They concluded that advanced life support skills improved outcome. Durham et al[28] also supported the use of prehospital intubation demonstrating that 58% of survivors of stab wounds and 67% of gunshot wound survivors underwent field intubation. This compared to 31% and 41%, respectively, among non-survivors. There were no survivors among the 30% of patients with blunt trauma who underwent field intubation. Fulton,[22] however, found 5 survivors among 189 (2.6%) who had prehospital intubation versus 1 survivor (1.8%) among patients intubated in the resuscitation suite.

Rosemurgy and colleagues[5] reported that more than 50% of their patients had scene times in excess of 20 minutes for intubation, intravenous access, extrication, and patient packaging. They had no survivors in their study, suggesting that advanced life support measures conveyed no advantage in their patients. Stratton et al[21] reported that none of the 3 neurologically intact patients with traumatic cardiopulmonary arrest who survived (all with penetrating trauma) had field intubation, despite that 83.1% of victims with penetrating trauma were intubated. Cera et al[7] found that field intubation had no impact on survival from traumatic cardiopulmonary arrest; it was performed in 53.8% of survivors and 73.4% of non-survivors. Perron et al[31] looked at prehospital intubation in pediatric patients and found that there was a significant reduction in mortality when comparing survival in all admitted patients (29%) versus those with prehospital intubation (19%).

Thus, the role of prehospital advanced life support measures remains controversial as it relates to the treatment of traumatic cardiopulmonary arrest. It is expected that field intubation may be helpful for primary etiologies with airway compromise such as hanging or high cervical injuries but may serve to prolong scene time and duration of CPR when performed to the exclusion of other measures. Support for intubation in the pediatric population is limited. Intravenous access is less well studied as it impacts outcome from traumatic cardiopulmonary arrest.

Air Medical Transport

The use of air medical transport for traumatic cardiopulmonary arrest has been examined by several authors. Wright and colleagues[32] evaluated the use of air transport in 67 trauma patients who sustained a cardiac arrest either before or during transport. In this group, there were 86.6% blunt trauma patients. Twenty patients (29.9%) were pronounced dead on-scene. Forty-seven were transported, of which only 6 responded with a pulse. All 47 patients died. Autopsies were available in 66 cases, 61 demonstrated major head injury, cervical spine injuries, or major thoracic injury. Rosemurgy et al[5] noted that 91% of patients in their study arrived via air medical transport. As noted above, there also were no survivors in their study. This supports the futility of air medical transport in such cases.

Falcone et al[11] reviewed 320 patients who had cardiopulmonary resuscitation following trauma. Within this group, 88.9% sustained

Table 3. Survival from Traumatic Cardiopulmonary Arrest for Pediatric Patients

AUTHOR	MECHANISM	NO. PATIENTS	NO. SURVIVAL	COMMENT
Sheikh et al[34]	Blunt	27	0 (0%)	Only included patients transported to hospital
Li et al[35]	Blunt, penetrating	957	224 (23.5%)	Included only patients taken to pediatric trauma center
Perron et al[31]	Blunt, penetrating	729	165 (22.6%)	Same database as Li et al
Suominen et al[36]	Blunt, penetrating	121	4 (3.3%)	All EMS trauma arrests
Fisher et al[37]	Blunt	65	1 (1.5%)	Only included patients transported to hospital
Calkins et al[38]	Blunt	25	2 (4%)	Only included patients transported to hospital

blunt injuries and 80% were transported directly from the scene of the injury. Only 6 of 320 (1.9%) patients survived. The average CPR time was 33 minutes. There was no advantage found for mechanism of injury, CPR time, or initial underlying rhythm. An analysis of the 6 survivors revealed that 3 were revived with bystander CPR (calling into question whether they were truly in cardiac arrest) and the 3 others had vital signs upon presenting to an outside emergency department. The recommendation was to use the initial response to resuscitation as a triage tool.

A protocol of using the response to initial resuscitation efforts to activate an air medical response was supported by Margolin and coworkers,[12] who found only 13 survivors among 67 patients who sustained traumatic cardiopulmonary arrest. Blunt trauma was the mechanism of injury in 79% of cases. Twelve of the 13 survivors had a sinus based rhythm on the transport team's arrival. Survivors also had a higher systolic blood pressure and revised trauma score on arrival of the air medical team. These authors concluded that response to initial resuscitation should dictate activation of the air medical team.

Lockey et al[24] described their 10-year experience in direct response to 909 cases of traumatic cardiopulmonary arrest. There were 68 (7.5%) survivors. Seven hundred forty (81.4%) were pronounced dead on-scene. The number of patients transported after resuscitation was 169, of which 131 were ultimately admitted to the hospital. It is unclear what percentage of these 169 patients had a response to resuscitation. These authors noted that patients with primary asphyxiating injury and penetrating trauma with cardiac tamponade had a better rate of survival.

Finally, DiBartolomeo and colleagues[33] compared outcomes between basic life support-staffed (BLS-staffed) ground EMS units and a physician-staffed helicopter in the outcomes of 129 patients who suffered traumatic cardiopulmonary arrest. The demographic distribution between the 56 patients treated by helicopter crews and 73 patients treated by ground crews were similar with regard to mechanism of injury and gender. There was a statistically but not clinically significant difference in age. There were no survivors treated by ground BLS response and 2 (3%) survivors of those treated by physician-staffed helicopter crew. Both patients were neurologically impaired. These authors suggested that larger scale studies would need to be performed to determine the efficacy of air medical response.

These studies suggest that the use of air medical transport is not futile but should be limited to patients who have a response to initial resuscitation on-scene or within the referring hospital.

Pediatric Patients

Although in general, age and gender have not been shown to be a factor in survival from traumatic cardiopulmonary arrest, it is reasonable to consider whether pediatric patients have a better outcome than adult patients. *(See Table 3.)* Sheikh and Brogan[34] reported on their 5-year experience with blunt trauma victims receiving both open and closed chest CPR. All patients were in cardiac arrest at the time of presentation. All but 4 of the patients were in asystole at the time of presentation to the trauma center, although 8 patients had PEA with a sinus rhythm when first treated by EMS. The mean CPR time was between 7 and 8 minutes. Despite that 15 patients received open thoracotomy at the time of presentation, no patient survived to discharge.

Pediatric traumatic cardiopulmonary arrest also was investigated by Li et al[35]who reported on 957 patients who underwent CPR either prior to or upon admission to pediatric trauma centers reporting to the National Pediatric Trauma Registry. Of these cases, there were 224 (23.5%) survivors. There was an almost universally dismal prognosis for patients with an asystolic rhythm on arrival (1 of 87 survived). Other factors that were associated with poor outcome included: systolic blood pressure less than 60 mmHg, comatose state, penetrating trauma, and need for CPR upon admission. Sixty-four percent of survivors had some functional disability. The data from the National Pediatric Trauma Database was further examined by Perron and colleagues,[31] who also reported survival in 25% of their study subjects. They confirmed poor outcome in patients with penetrating trauma and those children requiring additional CPR. In addition, prehospital intubation was associated with a worse outcome than that seen in those children who arrived unintubated. It should be remembered that both these results may be influenced by selection bias as out-of-hospital deaths as well as pediatric trauma arrests transported to non-trauma hospitals were not included.

Suominen et al[36] presented a more dismal picture in their 10-year review of pediatric traumatic cardiopulmonary arrest. They reviewed the outcomes of 41 pediatric patients with cardiac arrest, although 15 were secondary to smoke inhalation, strangulation or electrocution. Of the 26 remaining patients, 23 suffered a blunt injury and 3 suffered penetrating injury. Two of the 26 patients survived (7.7%), with only one who was neurologically intact. Only one of 21 patients whose initial rhythm was asystole survived. No trauma patient who arrived without vital signs survived.

Fisher and coworkers[37] described their 12-year experience

with 65 victims of blunt traumatic cardiopulmonary arrest in pediatric patients. Sixty-four (99%) of these patients expired, with 61 (93.8%) dying within the first 24 hours of admission. No patient survived neurologically intact. Of the 36 patients who had an autopsy, 31 had lethal cranial and cervical injuries, 16 had lethal abdominal injuries, and 12 had lethal thoracic injuries (myocardial rupture, aortic or vena caval disruption).

Most recently, Calkins and colleagues[38] described their experience with 25 children with traumatic cardiopulmonary arrest during a 3-year period. Sixty-four percent had CPR during transport, whereas 36% had CPR started in the resuscitation area. There were 2 survivors (8%), both of whom had CPR started in the trauma suite. Twenty-one patients died from severe head or spinal cord injury. Only 2 of 23 deaths (9%) were due to exsanguinating hemorrhage. Neither survivor had associated head injury or any neurologic sequelae on discharge.

These studies suggest that children have not been as well studied as adult populations but appear to have an equally dismal prognosis.

NAEMSP/COT Recommendations. Traumatic cardiopulmonary arrest, in general, has had a very dismal prognosis; this led the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma to develop a joint position paper[39] that discusses a rational and measured approach to patients who sustain a cardiac arrest after trauma. These recommendations attempt to incorporate many of the findings cited above and also have incorporated much of the literature addressing emergency thoracotomy (cited later in this text). The full recommendations can be found online at: *www.naemsp.org/pdf/guideterminate.pdf*. In short, the recommendations were offered to provide guidance to health care providers who attend to victims of traumatic cardiopulmonary arrest and define situations where such efforts may be futile, costly, and risky.

At least three studies have specifically evaluated the NAEMSP/COT recommendations. Pickens[13] compared the clinical circumstances for each of their 14 survivors and found that 13 would not have undergone resuscitation if their system had utilized these recommendations. They had two surviving blunt trauma victims who were pulseless, apneic, and without an organized rhythm on presentation. Two victims of penetrating trauma were initially in asystole. Three survivors had documented CPR times in excess of 15 minutes. Willis et al[23] noted that two of their 4 surviving patients who have qualified for termination of resuscitation efforts in the field had the NAEMSP/COT guidelines been utilized. Finally, Lockey et al[24] found that 13 (36%) of their 36 survivors would have breached the guidelines had they been strictly applied. This included 5 surviving blunt trauma victims who were pulseless, apneic, and without an organized rhythm; one patient with penetrating trauma who was apneic, pulseless and without signs of life; and 7 survivors whose cardiac arrest was witnessed and resuscitation efforts exceeded 15 minutes without response. Thus it appears that the current NAEMSP/COT recommendations are not 100% sensitive in identifying patients likely to fail resuscitation efforts.

Prehospital Care of Traumatic Cardiopulmonary Arrest

Based on the information provided above, it appears that there may be no absolute recommendations that can be made regarding the out-of-hospital disposition of patients with traumatic cardiopulmonary arrest. However, there are some treatment recommendations that can be made based on the likely causes of early trauma arrest and considering those conditions that may be potentially reversible. *(See Figure 1.)* These recommendations should be consistent with the treatment algorithms proposed in the International Trauma Life Support (ITLS), Prehospital Trauma Life Support (PHTLS), and Advanced Trauma Life Support (ATLS) courses. Continued support of the patient with traumatic cardiopulmonary arrest with chest compressions and other advanced cardiac life support is essential despite evidence that chest compressions themselves can be harmful.[25]

Airway with Cervical Spine Control and Ventilatory Assistance. Given that there are many causes of early death in which both the airway and cervical spine may be at risk, it seems reasonable to emphasize the establishment of a patent airway. Airway compromise from head injury, drug and alcohol intoxication, vomitus, and secretions, as well as direct airway injury, may lead to early death. Although airway compromise is the third leading cause of traumatic cardiopulmonary arrest behind craniocervical injury and thoracic vascular injury,[40] it must remain the first priority in approaching the patient.

Basic airway maneuvers should be emphasized first, including the jaw-thrust maneuver, neutral head positioning, and adequate suctioning of the airway. As demonstrated in the previous discussion, the role of prehospital endotracheal intubation in patients with traumatic cardiopulmonary arrest remains controversial. The discussion in these patients parallels recent concerns raised by other authors about the application of this skill in the out-of-hospital treatment of both adults[41,42] and children.[43] Prehospital intubation is probably most effective in the hands of providers who use it frequently, have continued training in this skill, and who are capable of completing this task quickly and competently.

The role of alternative airways such as the laryngeal mask airway, Combitube, pharyngeotracheal lumen airway, or newer single lumen airways (e.g., King LT) in this setting needs to be explored.

Restoration of Circulating Blood Volume. Since major thoracic vascular injury is a major cause of out-of-hospital death and thoracoabdominal hemorrhage is implicated in early in-hospital death, it is essential that intravenous access be established and fluid replacement be initiated to provide any hope of success.

With regard to vascular access, care must be taken to minimize the time on-scene in patients with traumatic cardiac arrest. The current recommendation for any trauma patient is to have two large bore intravenous catheters inserted with crystalloid solutions infusing as rapidly as possible. However, the benefit of this therapy must be weighed against the time consumed establishing vascular access.[44] Intravenous access should not delay transport to an ED, especially in an urban setting. When transport times are longer (rural settings), IV fluids become more important and a minimal delay in transport becomes less important in comparison.

Although there is significant debate about permissive hypotension particularly in the setting of penetrating trauma,[45] establishing a measurable blood pressure must remain a goal in the trauma arrest victim. At least one study has suggested that a systolic blood pressure of greater than 60 mmHg upon arrival to the hospital confers a survival advantage for victims of traumatic cardiopulmonary arrest.[28]

The current resuscitation fluid recommendation remains a crystalloid solution of either normal saline or lactated Ringer's solution. Blood substitutes may be a promising alternative in the future but recent trials have been hindered by ethical concerns.[46] The use of hypertonic saline has been explored in both military[47] and civilian[48] settings as a more efficient method of restoring circulating blood volume in the setting of early shock. However, there is not sufficient evidence to conclusively support the use of this agent in the routine management of profound blood loss or traumatic cardiopulmonary arrest.

Focusing on vascular access, intraosseous infusion remains a viable option that can be established rapidly[49] and provide for rapid infusion of virtually any resuscitation fluid at rates approaching large bore peripheral intravenous lines. Sites for insertion most commonly include the sternum and proximal femur; alternative sites, including the femur and proximal humerus, also have been used. The proximal tibia seems to be ideal since it is located away from the thorax where chest compressions are provided and the landmarks are easily identified. Lower extremity fractures may preclude the use of this site and complications such as compartment syndrome and osteomyelitis must be considered.

Other Interventions. In reviewing other potentially reversible causes of traumatic cardiac arrest, both tension pneumothorax and cardiac tamponade must be considered. The classic findings of tension pneumothorax, including distant or absent breath sounds, tympany on percussion of the involved hemithorax, neck vein distention, and tracheal deviation, may be difficult to appreciate in the out-of-hospital setting. The most reliable finding is difficult ventilation of these patients. Treatment of this condition in the out-of-hospital setting relies primarily upon needle decompression of the involved pneumothorax by insertion of a catheter into the second intercostal space. Some prehospital protocols will call for empiric needle decompression of both hemithoraces in all cases of traumatic cardiopulmonary arrest based on limitations in the physical assessment of these patients. Several case reports have noted that needle catheters were not effective in relieving a tension pneumothorax.[50,51] One prospective study addressed the effectiveness of this maneuver and concluded that needle decompression is of limited efficacy.[52] However, if accomplished quickly and effectively, there is essentially no added risk to the patient, who is already "dead" and has very little else to loose.

Several studies have noted that patients with pericardial tamponade have a favorable prognosis if identified early.[53-55] Here again, the classic signs of tamponade, including muffled heart tones and distended neck veins, may be difficult to appreciate in the prehospital setting. Decompression of cardiac tamponade using a subxyphoid approach has been taught in some prehospital texts and has been in use by EMS systems. However, data on the utility of this procedure in major cardiac injury indicate that it is not effective.[56,57] Contrary to needle decompression for suspected tension pneumothorax, a significant pericardial tamponade resulting in a cardiopulmonary arrest will almost certainly require a pericardiotomy for relief and control. In this instance, even a delay of a few moments can make the difference between life and death. If a cardiac tamponade is suspected and the patient is in an urban environment, transport should be the primary consideration.

Rapid Transport. There is no doubt that the duration of cardiac arrest has an impact both on patient mortality and long-term disability of survivors. In addition, the limited success of emergency thoracotomy depends on the timely delivery of a potentially salvageable patient. Each prehospital system should develop specific policies regarding the treatment and transport of patients with traumatic cardiopulmonary arrest based on their level of training, availability of providers with advanced skills such as air medical transport, proximity to the nearest emergency department, and proximity to the nearest trauma center. In urban environments, rapid transport with minimal intervention to the area trauma center is the most efficacious strategy. In more suburban environments, transport to the nearest hospital for stabilization and secondary transport to a trauma center may be the preferred strategy. Air medical support can be instrumental in accomplishing such secondary transfers.[12] The more challenging situations arise in rural locations where there is limited access to a trauma center, where transport times to the nearest hospital can be prolonged and where the provider may be capable of basic life sup-

Figure 1. Prehospital Treatment Guidelines for Traumatic Cardiopulmonary Arrest

ENVIRONMENT	TREATMENT GUIDELINES
Urban	Basic airway and spine protection measures Definitive airway and intravenous access enroute Consider needle decompression of chest Rapid transport — trauma center preferred
Suburban	Basic airway and spine protection measures Consider definitive airway and intravenous access Fluid resuscitation Needle decompression of chest Consider pericardiocentesis (if allowed by protocol) Rapid transport — nearest hospital
Rural	Consider air medical transport for ALS skills Definitive airway and intravenous access at scene Fluid resuscitation Needle decompression of chest Consider pericardiocentesis (if allowed by protocol) Rapid transport to nearest hospital or consider air medical transport for any response

Figure 2. Treatable Conditions Leading to Traumatic Cardiopulmonary Arrest

CONDITION	TREATMENT
Airway obstruction/compromise	Establish airway
Hypoventilation (spinal cord injury, ruptured diaphragm)	Ventilate
Open pneumothorax	Seal/ventilate
Tension pneumothorax	Decompress
Massive hemothorax (major vascular or cardiac injury)	Resuscitate/control*
Cardiac tamponade	Decompress/repair
Hemoperitoneum (solid organ or vascular injury)	Resuscitate/control
Retroperitoneal hemorrhage	Resuscitate/control
Non-torso exsanguination	Resuscitate/control

* Control of hemorrhage should parallel resuscitation, and will most often require surgical intervention in the unstable patient.

port only. Options in such conditions include use of air medical support to provide advanced procedures; airway stabilization with CPR and advanced life support intercept; and field pronouncement after a period of airway intervention and cardiac compressions. The specific option chosen should be addressed in regional trauma triage protocols.

Hospital Care of Traumatic Cardiopulmonary Arrest

Care of the patient with traumatic cardiopulmonary arrest should be based on the tenets of ATLS, with a focus on the identification and treatment of immediately correctable causes of cardiac arrest. *(See Figure 2.)* Treatment priorities would include establishment of a definitive airway, provision of adequate ventilation, and insertion of chest tubes in patients with tension pneumothorax, massive hemothorax, bronchial or esophageal disruption, or diaphragmatic tear. The patient's volume status should be addressed with the administration of crystalloid solution through large bore peripheral intravenous catheters. Larger infusion catheters may be utilized and early autologous and allogeneic blood transfusion also can be considered. Either a FAST exam or diagnostic peritoneal lavage should be performed to diagnose massive hemoperitoneum. The focused abdominal sonography in trauma or FAST examination has the advantage of providing information about possible pericardial tamponade and cardiac injury. Immediate radiographs of the pelvis should be obtained to detect massive retroperitoneal hemorrhage.

However, the major decision that must be made in cases of traumatic cardiopulmonary arrest is whether to perform an emergency thoracotomy. This procedure was originally described in 1897 by Rehn[58] and was rediscovered for management of traumatic cardiac arrest in the 1960s.[59,60] The procedure is performed using a left anterior lateral approach after the patient's chest has been rapidly prepped. In about 20% of cases,[61] the incision will require an extension across the right chest to control hemorrhage. The major steps involved once the chest has been opened include: 1) direct control of any cardiac or vascular injuries; 2) identification and relief of pericardial tamponade via pericardiotomy; 3) maintenance of major organ perfusion via cross-clamping of the thoracic aorta; 4) maintenance of forward blood flow by open cardiac compression; and 5) cross-clamping the pulmonary hilum to control of air embolism. None of these subprocedures are easy. They all require training and practice. A discussion of the technique of ED thoracotomy and the credentialing of the provider are beyond the scope of this paper. Both, however, should be discussed and decided in advance.

Outcomes studies on emergency thoracotomy have been hampered by the same problems that have beset studies of prehospital cardiopulmonary arrest. They include a lack of uniform definitions and endpoints, as well as selection bias for patients taken to trauma centers. More specifically, emergency thoracotomy has been studied in a variety of disparate situations, including not only those procedures performed on the patient arriving without a pulse, but also those who lose a pulse either in the emergency department or operating suite, those who remain persistently hypotensive despite adequate resuscitation, and even those who undergo thoracotomy for ongoing thoracic hemorrhage.

The most comprehensive review on this subject was conducted by Rhee and colleagues[62] in 2000. These authors considered only studies in which both penetrating and blunt trauma were included. Their analysis focused on true emergent thoracotomies defined as those performed in the emergency department or trauma resuscitation area. The authors examined the factors that contributed to a successful discharge from the hospital and normal neurologic outcome. Twenty-four studies from 1974 through 1998 were included in their meta-analysis. The overall survival rate was 7.4% for the 4620 thoracotomies reported in this study. The survival range was from 1.8% to 27.5% depending on patient selection. Nine of the 24 studies had survival rates in excess of 10% of eligible patients. These authors demonstrated survival rates of 1.4% for blunt trauma victims, 4.3% for gunshot wounds, and 16.8% for stab wounds. Isolated cardiac injury had a survival rate of 19.4%, whereas patients with multiple injury sites had a survival of 0.7%.

The data also were examined for patients who lost signs of life, defined as cardiac electrical activity (organized cardiac rhythm), respiratory effort, or pupillary response. Patients who lost signs of life in the field had a survival of 1.2% compared to 8.9% for those who arrested during transport or 11.5% who demonstrated signs of life on arrival and subsequently arrested during resuscitation.

Although neurologic outcome was not routinely reported, survivors were neurologically normal 92.7% of the time when reported. Based on Rhee and coworkers'[62] analysis, the following recommendations for emergency thoracotomy were made: 1) it should be performed for victims of penetrating trauma with signs of life in the field not responding to fluid resuscitation or those who lose vital signs during resuscitation; 2) it should be performed for patients with penetrating injury with any sign of life in the field who is in traumatic cardiopulmonary arrest on arrival;

3) it should be performed for patients with blunt trauma who lose signs of life in the resuscitation area or immediately prior to arrival (not further defined); and 4) no emergency thoracotomy be attempted on patients who arrive without signs of life who did not have signs of life in the field. The authors did acknowledge, however, that there is no uniform predictor of mortality.

Fialka et al[29] supported the recommendation regarding performance of an emergency thoracotomy in blunt trauma victims who lose signs of life immediately before arrival. They reported 4 survivors out of 38 blunt trauma patients who had CPR for between 11 and 15 minutes prior to thoracotomy. Only one of these survivors had signs of life on arrival to the resuscitation area. Coats et al[48] confirmed that only penetrating victims with signs of life at the scene are amenable to emergency thoracotomy. There were no survivors among their patients with penetrating injury who did not demonstrate any signs of life on-scene.

More recently, Powell et al[63] shed further light on these recommendations. They reported on 26 survivors among 959 patients who underwent emergency thoracotomy. Only 4 survivors sustained blunt trauma and the maximum duration of prehospital CPR was 5 minutes. No survivor of penetrating trauma had prehospital CPR of more than 15 minutes. These authors reported 5 survivors with an initial rhythm of asystole which had been thought to be a uniformly poor prognostic indicator. Each of these patients had pericardial tamponade. Kennedy and Sharif[64] reviewed their personal experience in performing over 100 emergency thoracotomies. They reported that only 7 survived to discharge. All were victims of penetrating injury and no survivors were in traumatic cardiopulmonary arrest upon arrival of paramedics. This author has also advocated against the use of abdominal cross-clamping in performing an emergency thoracotomy and withholding the procedure for those in cardiac arrest for more than 20 minutes. Finally, Sheppard et al[65] advocated for one additional indication for emergency thoracotomy: traumatic cardiopulmonary arrest from non-torso injury. In their series of 959 patients undergoing emergency thoracotomy, 27 had non-truncal trauma. Three of 27 (11%) survived.

Pediatric patients who undergo emergency thoracotomy have demonstrated mixed results. Beaver et al[66] had no survivors among 15 children with blunt trauma patients and 2 with penetrating trauma who underwent emergency thoracotomy. Powell and colleagues,[67] however, demonstrated 4 survivors (36%) among 11 children with penetrating injury and cardiac arrest as well as one survivor (12.5%) among the 8 blunt injury victims.

These results suggest that ideally emergency thoracotomy should be performed within 5 minutes of losing vital signs for victims of blunt trauma and 15-20 minutes for victims of penetrating trauma. Special consideration should be given to victims of non-torso trauma. Emergency thoracotomy should not be undertaken for trauma victims who had no signs of life in the field and have not responded to resuscitative efforts both in the field or following initial assessment in the resuscitation area. *(See Figure 3.)*

One special consideration that must be discussed is the care of the pregnant female who has sustained a traumatic cardiopulmonary arrest. In this setting, a rapid decision must be made with regard to performing a perimortem cesarean section. The decision should be based on gestational age and fetal viability ex-utero, and the duration of cardiopulmonary arrest. Katz and coworkers[68] first introduced the term "perimortem cesarean section" and suggested that there was a 4-minute limit to fetal viability. This short interval was determined based on the limited cardiac output produced during CPR in pregnancy due to caval compression by the uterus. Since that time, there have been multiple case reports of viable infants being delivered in the perimortem period.[69,70] A recent series from Katz et al[71] described 38 cases of perimortem cesarean section in the literature from 1986-2004. The leading cause of death was maternal trauma. The authors suggest that the period of viability extends far beyond the 4 minute limit. In fact, 7 of 38 cases described had the procedure performed after 15 minutes of CPR and the infant was neurologically intact in 4 cases. This suggests that although the decision to perform an emergent perimortem cesarean section must be made rapidly, the previous limit of 4 minutes to perform the procedure appears to be arbitrary.

Figure 3. Recommendations for Performing an Emergency Thoracotomy

1. **Blunt Trauma:** Emergency thoracotomy should be performed within 5 minutes of vital sign loss.
2. **Penetrating Trauma:** Emergency thoracotomy should be performed within 15 to 20 minutes of vital sign loss.
3. **Non-torso Trauma:** Special consideration should be given to performing an emergency thoracotomy in victims with cardiac arrest.
4. **No Signs of Life in the Field:** Emergency thoracotomy should not be undertaken for trauma victims who have not responded to resuscitative efforts either in the field or following initial assessment in the resuscitation area.

Conclusion

Almost 80% of trauma deaths occur in the first few hours following injury. Therefore, it is likely that many prehospital, emergency department, and general surgery practitioners will encounter patients with traumatic cardiac arrest in their practices. Although neurologically intact survival is rare, survival from traumatic cardiopulmonary arrest is not unknown. Studies have shown that the resources dedicated to the care of these patients and the costs incurred may be offset by the return in productivity achieved by these rare survivors.

Prehospital providers must be aware of their treatment options and limitations in caring for victims of traumatic cardiopulmonary arrest. The closer in proximity one is to a trauma center, the less intervention there is to be performed on-scene. Rural providers are hindered by longer transport times and limited advanced skills. Air medical teams should be considered a potential resource in this environment.

In the hospital, initial efforts should be focused on establishing a definitive airway and intravenous access and on identifica-

tion of immediately treatable conditions. Successful resuscitation must be coupled with hemorrhage control. A rapid decision must be made regarding the benefit of emergency thoracotomy. Blunt trauma patients with more than 5 minutes of CPR and penetrating trauma victims with more than 15 minutes of CPR are unlikely to survive. The pregnant trauma patient with a viable fetus should be considered for a perimortem C-section.

Although victims of traumatic cardiopulmonary arrest with penetrating trauma, signs of life on arrival to the resuscitation area, and an underlying non-perfusing sinus rhythm have a better outcome, there are no absolute predictors of survival.

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CNE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CNE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a letter of credit.** When your evaluation is received, a letter of credit will be mailed to you.

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CME/CNE Questions

1. Major causes of prehospital trauma deaths include:
   A. major head injury.
   B. Spinal cord injury.
   C. traumatic aortic rupture.
   D. All of the above

2. A patient with which of the following initial cardiac rhythms is least likely to survive?
   A. PEA
   B. Ventricular fibrillation
   C. Asystole
   D. Sinus tachycardia

3. Shorter duration of CPR is associated with better survival, but prolonged CPR does not preclude a successful outcome.
   A. True
   B. False

4. Which of the following is true regarding ALS measures for prehospital traumatic cardiopulmonary arrest?
   A. Survivors were more likely to have successful endotracheal intubation and intravenous access.
   B. Long scene times led to better outcomes.
   C. All pediatric patients in traumatic cardiopulmonary arrest should be intubated.
   D. The value of field intubation is clear.

5. Studies suggest that the use of air medical transport is not futile, but should be limited to patients who have a response to initial resuscitation on-scene or within the referral hospital.
   A. True
   B. False

6. Pediatric traumatic cardiopulmonary arrest has been well studied and is associated with a better outcome than that found with adults.
   A. True
   B. False

7. The current resuscitation fluid of choice is:
   A. blood substitutes.
   B. crystalloid solution.
   C. hypertonic saline.
   D. colloids.

8. Which of the following is a recommended treatment guideline in a rural setting?
   A. Consider air medical transport for ALS skills
   B. Definitive airway and intravenous access at the scene
   C. Fluid resuscitation
   D. All of the above

9. Which of the following is a recommendation for performing emergency thoracotomy?
   A. Emergency thoracotomy should be performed within 45 minutes of vital sign loss for blunt trauma patients.
   B. Emergency thoracotomy should be performed within 15-20 minutes of vital sign loss for penetrating trauma patients.
   C. Emergency thoracotomy should be performed on patients who do not respond to resuscitative efforts in the field.
   D. Emergency thoracotomy should be performed on patients who do not respond to resuscitative efforts in the initial assessment in the ED.

10. Which of the following is true regarding the care of the pregnant trauma patient in cardiopulmonary arrest?
   A. Perimortem C-sections should never be performed.
   B. The decision to perform a perimortem C-section should consider gestational age and fetal viability.
   C. The leading cause of fetal death is direct fetal trauma.
   D. A perimortem C-section should never be considered after 4 minutes.

Answers: 1. D; 2. C; 3. A; 4. A; 5. A; 6. B; 7. B; 8. D; 9. B; 10. B

In Future Issues:

Pitfalls in Trauma Management

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Upon completing this program, the participants will be able to:

1. discuss conditions that should increase suspicion for traumatic injuries
2. describe the various modalities used to identify different traumatic conditions
3. cite methods of quickly stabilizing and managing patients
4. identify possible complications that may occur with traumatic injuries

Each issue of your newsletter contains questions relating to the information provided in that issue. After reading the issue, answer the questions at the end of the issue to the best of your ability. You can then compare your answers with the correct answers provided in an answer key in the newsletter. If any of your answers were incorrect, please refer back to the source material to clarify any misunderstanding.

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