CME CREDIT

NADJA G. PETER, MD
Clinical Associate in Pediatrics, Craig-Dalsimer Division of Adolescent Medicine, The Children's Hospital of Philadelphia

LIANA R. CLARK, MD
Assistant Professor of Pediatrics, Craig-Dalsimer Division of Adolescent Medicine, The Children's Hospital of Philadelphia

JEFFREY R. JAEGER, MD
Assistant Professor of Medicine, Division of General Internal Medicine, University of Pennsylvania Health System, Philadelphia

Fitz-Hugh-Curtis syndrome: A diagnosis to consider in women with right upper quadrant pain

■ ABSTRACT

Fitz-Hugh-Curtis syndrome—inflammation of the liver capsule associated with genital tract infection—occurs in up to one fourth of patients with pelvic inflammatory disease (PID). Classically presenting as sharp, pleuritic right upper quadrant pain, usually but not always accompanied by signs of salpingitis, it can mimic many other common disorders such as cholecystitis and pyelonephritis.

■ KEY POINTS

Neisseria gonorrhoeae and *Chlamydia trachomatis* are thought to be the primary causative agents of Fitz-Hugh-Curtis syndrome.

The pathogenesis of Fitz-Hugh-Curtis syndrome is incompletely understood. It may result from direct, hematogenous, or lymphatic infection of the liver capsule and related structures, or from an exaggerated immune response to *C trachomatis*.

The incidence ranges from 4% to 14% in women with PID, but is as high as 27% in adolescents with PID, whose less-mature anatomy makes them more susceptible to infection.

The diagnosis is usually made clinically by eliminating other causes of right upper quadrant pain and isolating the pathogen. This can be difficult if salpingitis is absent.

Treatment consists of antibiotics directed against *N gonorrhoeae* and *C trachomatis*; mechanical lysis of adhesions can be performed laparoscopically if conservative treatment fails.

F ITZ-HUGH-CURTIS SYNDROME—perihepatitis associated with pelvic inflammatory disease—can pose a diagnostic challenge to the clinician, especially when right upper quadrant pain predominates, mimicking acute gall bladder disease. This article reviews the etiology, pathogenesis, diagnosis, and treatment of this syndrome.

■ HISTORICAL PERSPECTIVE

Association with gonorrhea

Fitz-Hugh-Curtis syndrome was first described in 1920 by Carlos Stajano,[1] who noted adhesions between the liver capsule and anterior abdominal wall in patients with gonococcal infection and right upper quadrant pain.

In the 1930s Thomas Fitz-Hugh and Arthur Curtis also described the syndrome and made the connection between the acute clinical syndrome of right upper quadrant pain following a pelvic infection and the "violin-string" adhesions found in women with evidence of prior salpingitis.[2,3] Curtis described several cases of these very typical adhesions between the liver and the abdominal wall in patients with gonococcal disease and noted that similar adhesions were not found in other causes of peritonitis, suggesting the combination was a unique syndrome.[4] Fitz-Hugh suggested that *Neisseria gonorrhoeae* was the cause when he found gram-negative diplococci on smears from the liver capsule in patients with the syndrome.[2]

Since then, the diagnosis of Fitz-Hugh-Curtis syndrome has largely been a clinical

one, based on the history and physical examination plus positive cultures. As the syndrome became better known, it began to be diagnosed more frequently during surgical exploration for other problems such as infertility or presumed cholecystitis. Laparoscopy further improved the ability to detect the syndrome and is an option when lysis of adhesions is necessary to relieve its symptoms.

Chlamydia also implicated

For many years, *N gonorrhoeae* was thought to be the sole causative agent of the syndrome.[2,5,6] In 1978, however, Muller-Schoop et al[7] first demonstrated serologic evidence of acute infection with *Chlamydia trachomatis* in 9 of 11 patients who had undergone laparoscopic evaluation for peritonitis, 6 of whom also had perihepatitis. Others have since documented similar findings.[8,9]

C trachomatis has also been cultured from the cervix, the fallopian tubes, and in a few cases from the liver capsule in patients with perihepatitis.[10,11] Most experts now believe *C trachomatis* is the culprit more often than *N gonorrhoeae* and is the likely explanation for most "culture-negative" cases described before *C trachomatis* infection was recognized as a sexually transmitted disease.[9]

■ HOW COMMON IS FITZ-HUGH-CURTIS?

Estimates of the incidence of Fitz-Hugh-Curtis syndrome depend on the diagnostic criteria used, as patients with no symptoms sometimes have impressive perihepatic adhesions seen at laparoscopy, while patients with pelvic inflammatory disease (PID) and right upper quadrant pain may have no laparoscopic evidence or other signs of perihepatitis.

Using only clinical criteria, Semchyshyn[12] found perihepatitis in 12% of patients with PID. Onsrud[13] used laparoscopic criteria and found a similar (13.8%) rate of coincident perihepatitis and PID. In this study, which examined all cases of PID seen in the gynecologic department of one hospital in 2 years, 37% of the patients with laparoscopic evidence of both PID and perihepatitis had no right upper quadrant symptoms. The incidence of laparoscopic perihepatitis was higher in women who had undergone placement of an intrauterine con-

traceptive device (IUD) within the previous 6 weeks than in those who had had such a device for a longer period.

Others have found lower rates of Fitz-Hugh-Curtis syndrome. In a study in Sweden between 1978 and 1982, Paavonen et al[8] found perihepatitis in 4% of 322 women with laparoscopic evidence of PID. Wang et al[9] reported that only 17 (3.8%) of 442 women referred for evaluation of PID had clinical evidence of Fitz-Hugh-Curtis syndrome.

Rates higher in adolescents

In contrast, Litt and Cohen[6] found clinical evidence of perihepatitis (right upper quadrant tenderness or elevated liver enzymes) in 37 (27%) of 137 adolescents with salpingitis.

The high rate in this study may be partially explained by anatomical features of the adolescent genitourinary tract that facilitate progression of cervicitis to PID and, presumably, to Fitz-Hugh-Curtis syndrome. For example, the transitional zone between squamous and columnar epithelium of the cervix (the ectropion) is at the outer margin of the cervix in adolescents, vs within the cervix in adults.[14]

■ PATHOGENESIS IS UNCERTAIN

The pathogenesis of the Fitz-Hugh-Curtis syndrome is still poorly understood. Several mechanisms have been proposed.

Direct infection of the liver?

Traditionally, inflammation of the liver capsule has been attributed to direct bacterial infection.[4] Organisms were thought to travel from the genital area via the fallopian tubes and the paracolic gutters to the liver capsule.[4,15]

Some evidence supports this: Holm-Nielsen et al[16] demonstrated that peritoneal fluid is propelled from the pelvis to the diaphragm, where it is preferentially absorbed on the right side. The association between recent insertion of an IUD and Fitz-Hugh-Curtis syndrome[13] also supports the hypothesis of intraperitoneal spread of organisms.

While direct spread may occur in some cases, several factors suggest an alternate etiology. Bacteria have only rarely been isolated from the liver surface or surrounding ascites in patients with Fitz-Hugh-Curtis syndrome.[5,11]

If Fitz-Hugh-Curtis syndrome is present, screen for other STDs, including HIV

Also, if organisms traveled via the peritoneum, we should see inflammation and infection of structures between the pelvis and the liver, but evidence of this is rare.[17,18] In addition, Fitz-Hugh-Curtis syndrome has been reported in men, in whom a mechanism other than direct infection must exist.

Hematogenous spread?

Could bacteria travel from the pelvis to the liver via the blood stream? The theory is supported by a case report[19] that found focal lesions in the liver of a patient with Fitz-Hugh-Curtis syndrome, which resolved following antibiotic therapy. However, there is no evidence to support disseminated blood infection in most cases.

Lymphatic spread?

It is plausible that bacteria could spread from the pelvis to the liver capsule through the lymphatic system, and this mechanism would explain why most patients with Fitz-Hugh-Curtis syndrome show no evidence of generalized intra-abdominal infection or disseminated blood stream infection. However, most of the lymphatic drainage in the female reproductive tract is retroperitoneal, with no anatomical evidence linking the pelvic and subdiaphragmatic lymphatic systems.[17]

An exaggerated immune response?

Perihepatitis and Fitz-Hugh-Curtis syndrome may represent a "hyperimmune" response to C trachomatis.[20,21] Several studies[9,20–22] demonstrated higher serum titers of antichlamydial immunoglobulin G (IgG) antibodies in patients with perihepatitis and salpingitis than in patients with salpingitis alone.

There is evidence that variable host factors can affect the inflammatory response to Chlamydia infection: for example, postinfectious scarring of the eyelid due to C trachomatis is more common in patients with certain HLA class I antigens.[22] Patton et al[23] produced perihepatitis in a pig-tail macaque after exposing it to one strain of C trachomatis and then rechallenging it with different strains, suggesting that the vigorous immune response manifested by perihepatitis might be a reinfection phenomenon. Why such a response should be limited to the liver capsule is unknown.

Certain aspects of the organism may also influence the host response. Recent attention has focused on the Chlamydia 60-kd heat-shock protein Chsp60, which exhibits considerable homology with human heat-shock proteins.[24] An immune response triggered by C trachomatis might cross-react with human heat-shock proteins in other tissues.

Money et al[20] compared 27 patients with laparoscopically diagnosed perihepatitis and salpingitis and 46 patients with salpingitis alone. Elevated levels of antibody to Chsp60 were found in 67% of the perihepatitis-salpingitis group vs only 28% of the salpingitis-alone group. The median titer of the Chsp60 antibody was also significantly higher in the perihepatitis-salpingitis group. Antichlamydial IgG and IgM levels were not significantly different between the two groups. The authors concluded that the Chsp60 antigen influences the host's inflammatory response. However, whether Chsp60 is involved in inducing immunopathology or whether it is a byproduct of severe infection remains unclear.[25]

■ DIAGNOSIS IS DIFFICULT

Fitz-Hugh-Curtis syndrome can be difficult to diagnose, as its symptoms and physical findings can mimic those of many other diseases (**TABLE 1**). It is most often mistaken for acute cholecystitis, especially in cases in which the right upper quadrant pain is more pronounced than the pelvic symptoms, or when the perihepatitis presents long before or after the symptoms of PID.[26]

Perihepatitis can be definitively distinguished from other causes of right upper quadrant pain only by directly visualizing the liver by laparoscopy or laparotomy.[6,27] However, in the right clinical setting, the diagnosis can be adequately established by excluding other causes and isolating a characteristic pathogen.[28]

Symptoms

Symptoms of acute or subacute PID (fever, abdominal pain, vaginal discharge) are almost always present.[29]

Right upper quadrant pain. The perihepatic component usually presents as sharp pleuritic pain localized to the right upper quadrant at the lower rib margin, likely relat-

Most experts now believe C trachomatis is the culprit more often than N gonorrhoeae

TABLE 1

Differential diagnosis of Fitz-Hugh-Curtis syndrome

Cholelithiasis
Cholecystitis
Pleurisy
Pneumonia
Pulmonary embolism
Rib fracture
Pyelonephritis
Hepatitis
Nephrolithiasis
Perforated ulcer
Subphrenic abscess
Pancreatitis
Appendicitis
Herpes zoster
Enteroviral epidemic pleurodynia
  (Bornholm disease)

Ultrasound is most useful in excluding other common causes of right upper quadrant pain

ed to inflammation of the underside of the diaphragm.[2,17] The pain may be referred to the right shoulder or to the inside of the right arm and may be accompanied by nausea, vomiting, hiccupping, chills, fever, night sweats, headache, and malaise.[17] Movement often exacerbates the pain.[2]

The right upper quadrant pain may follow the lower abdominal pain by days, or the two may occur simultaneously. Rarely, the right upper quadrant pain is the presenting symptom without lower abdominal pain.[7,26,28,30] This occurs in patients who have recovered from an acute episode of PID without appropriate treatment. In this more indolent presentation, the pain may be due to adhesions between the liver capsule and the diaphragm or the abdominal wall.[17]

Although most patients present with right-sided pain, a few cases of left upper quadrant pain with perisplenitis seen on laparoscopy have also been reported.[31]

Physical findings

On physical examination, patients have moderate to severe tenderness in the right upper quadrant, with some guarding and possibly splinting.

A friction rub may be heard along the right anterior costal margin. (Fitz-Hugh described it as "beautiful 'new snow' creaking frictions."[2])

A bimanual pelvic examination may detect vaginal discharge, cervical motion tenderness, or adnexal tenderness previously unnoticed by the patient. These findings suggest underlying PID.

Identifying the pathogen

The pathogen is most commonly isolated from a cervical specimen, but if clinical suspicion is high, rectal, urethral, and pharyngeal samples should be obtained as well.

There are several tests for C trachomatis and N gonorrhoeae. Cultures are still widely used, but genetic amplification tests such as the ligase chain reaction (LCR) and nucleic acid amplification test are highly sensitive and specific, making them promising for diagnosing both N gonorrhoeae and C trachomatis. They can be performed on vaginal, urine, and cervical samples.[14] Their main limitation is their cost. Serologic tests specific for C trachomatis can also be helpful.[14,32]

Radiographic studies

Radiographic studies are most useful to rule out other possible causes.

Chest and abdominal radiographs may exclude pneumonia or free air under the diaphragm.

Ultrasonography is the study of choice for evaluating the gallbladder and liver, and can exclude cholecystitis, cholelithiasis, and other common causes of right upper quadrant pain. It can also help evaluate the ovaries for abscesses or other findings consistent with PID.

In addition, typical ultrasonographic abnormalities in the perihepatic area have been detected in patients with Fitz-Hugh-Curtis syndrome[19,33–35]:

• Dinerman et al[33] described an adolescent with PID not responding to usual treatment who had ultrasonographic evidence of ascitic fluid in the hepatorenal space and at the splenic hilus.

• Romo and Clarke[34] described a patient with Fitz-Hugh-Curtis syndrome who had large amounts of loculated fluid in the abdomen and pelvis, seen by both ultrasonography and computed tomography.

- Van Dongen[35] described two patients in whom ascites and adhesions between the liver capsule and the abdominal wall were clearly seen on ultrasonography.
- Schoenfeld et al[36] found that the right anterior extrarenal space (measured by ultrasonography) was wider in 9 patients with Fitz-Hugh-Curtis syndrome than in 72 patients without clinical signs of the syndrome, suggesting that such a finding should prompt a search for genital tract infection.

Further study is needed to confirm these findings and evaluate the usefulness of these tools in diagnosing Fitz-Hugh-Curtis syndrome. Clinically, ultrasonography is most useful in excluding more common causes of right upper quadrant pain (TABLE 1).

Computed tomography. Contrast enhancement of the liver capsule can also support the diagnosis.[37]

Laboratory tests may provide clues
Laboratory tests are only partially helpful with Fitz-Hugh-Curtis syndrome.

Liver enzyme levels are usually normal or only slightly elevated, which can help rule out hepatitis.[5,20,28,38] Litt and Cohen,[6] in a 1978 case series, cited a high rate of nonspecific elevations in transaminase levels in patients with Fitz-Hugh-Curtis syndrome. The enzyme abnormalities responded to antibiotics, suggesting that the perihepatic inflammation was responsible for the abnormal levels.

The erythrocyte sedimentation rate (ESR) has conflicting evidence for its use in the diagnosis of Fitz-Hugh-Curtis syndrome. Some small series and case reports demonstrated elevated ESRs in patients with Fitz-Hugh-Curtis syndrome,[10,19,28,38] but larger studies have found a less robust association.[6,20] Miettinen at al[39] demonstrated that ESR elevation could distinguish mild from severe PID, but their cohort included only 5 patients with perihepatitis.

The white blood cell count may be normal or elevated.[39]

Surgical exploration
Surgical exploration is warranted only if symptoms do not resolve with therapy. Laparoscopy can help confirm the diagnosis of PID in patients with suspected Fitz-Hugh-Curtis syndrome, as well as help exclude other diagnoses.[27,40]

Counseling and further testing
The diagnosis of Fitz-Hugh-Curtis syndrome provides an opportunity for education and counseling about safer sex, and should prompt the clinician to screen for other sexually transmitted diseases including human immunodeficiency virus (HIV) infection.

■ TREATMENT SIMILAR TO PID

The management of Fitz-Hugh-Curtis syndrome is similar to that of PID.[41,42]

Most patients can be treated as outpatients, although hospitalization should be strongly considered if the patient is:
- Adolescent (a group whose anatomy and high rate of noncompliance put them at particularly high risk for reproductive complications[14,32,41])
- Pregnant
- Immunodeficient
- A potential candidate for surgery (eg, if cholecystitis cannot be excluded)
- Unreliable for follow-up[32,39]
- Having particularly severe symptoms
- Unresponsive to oral therapy or unable to tolerate oral medication.[41,42]

Antibiotic therapy
Antibiotics should be directed at the most likely pathogens, in particular *N gonorrhoeae*, *C trachomatis*, facultative gram-negative rods, and anaerobes,[14,30] since isolation of all offending agents is unlikely.

The Centers for Disease Control and Prevention has recently issued guidelines for treating PID (TABLE 2). If the patient is on parenteral therapy, it should be continued for 48 hours after she shows clinical improvement; then oral therapy can be started to complete 14-days.[41–43]

The symptoms of perihepatitis usually resolve quickly once appropriate treatment is started, which in difficult cases may lend support to the diagnosis.

Managing complications
Long-term complications of Fitz-Hugh-Curtis syndrome are rare and can mostly be attrib-

Management is similar to that of pelvic inflammatory disease

TABLE 2

CDC guidelines for treating pelvic inflammatory disease

Parenteral regimens

Either **cefotetan** (Cefotan) 2 g intravenously (IV) every 12 hours or **cefoxitin** (Mefoxin) 2 g IV every 6 hours; plus

Doxycycline (Doryx and others) 100 mg orally (PO; preferred) or IV every 12 hours; followed by

Doxycycline 100 mg PO twice a day to complete 14 days

Clindamycin (Cleocin) 900 mg IV every 8 hours; plus

Gentamicin (Garamycin) in a loading dose of 2 mg/kg of body weight IV or intramuscularly

(IM) followed by 1.5 mg/kg every 8 hours (single daily dosing may be used); followed by

Either **doxycycline** 100 mg PO twice a day or **clindamycin** 450 mg PO four times a day to complete 14 days

Alternative parenteral regimens

Either **ofloxacin** (Floxin) 400 mg IV every 12 hours or **levofloxacin** (Levaquin) 500 mg IV once daily; with or without

Metronidazole (Flagyl and others) 500 mg IV every 8 hours*; followed by

Doxycycline 100 mg PO twice a day to complete 14 days

Ampicillin/sulbactam (Unasyn) 3 g IV every 6 hours; plus

Doxycycline 100 mg PO or IV every 12 hours; followed by

Doxycycline 100 mg PO twice a day to complete 14 days

Oral regimens

Either **ofloxacin** 400 mg PO twice a day for 14 days or **levofloxacin** 500 mg PO once daily for 14 days; with or without

Metronidazole 500 mg PO twice a day for 14 days*

Either **ceftriaxone** (Rocephin) 250 mg IM in a single dose, **cefoxitin** 2 g IM in a single dose and **probenecid** (Benemid, Probalan) 1 g PO given concurrently in a single dose, or another parenteral third-generation cephalosporin (eg, **ceftizoxime** or **cefotaxime**); plus

Doxycycline 100 mg PO twice a day for 14 days; with or without

Metronidazole 500 mg PO twice a day for 14 days

---

*Ofloxacin alone has been demonstrated to be effective; however, concerns about its effectiveness against anaerobes have led to the addition of metronidazole to fluoroquinolone-alone regimens

ADAPTED FROM CENTERS FOR DISEASE CONTROL AND PREVENTION. SEXUALLY TRANSMITTED DISEASES TREATMENT GUIDELINES 2002. MMWR 2002; 51(RR-6):1–78.

Several regimens are possible

uted to the associated PID. Complications of PID include pain and small bowel obstruction from adhesions, as well as infertility.

If pain persists despite adequate treatment, a prompt laparoscopic examination should be performed to determine whether there are adhesions in the perihepatic area or elsewhere.[44] Laparoscopy provides less invasive therapy than laparotomy when lysis of adhesions is necessary for symptom relief. ❧

■ REFERENCES

1. **Stajano C.** La reaccion frenica en ginecologica. Semana Medica-Beunoa Airea 1920; 27:243–248.
2. **Fitz-Hugh T Jr.** Acute gonococcic peritonitis of the right upper quadrant in women. JAMA 1934; 102:2094–2096.
3. **Curtis A.** A cause of adhesions in the right upper quadrant. JAMA 1930; 94:1221–1222.
4. **Curtis A.** Adhesions of the anterior surface of the liver. JAMA 1932; 99:2010–2012.
5. **Kornfeld SJ, Worthington MG.** Culture-proved Fitz-Hugh-Curtis syndrome. Am J Obstet Gynecol 1981; 139:106–107.
6. **Litt IF, Cohen MI.** Perihepatitis associated with salpingitis in adolescents. JAMA 1978; 240:1253–1254.
7. **Muller-Schoop JW, Wang SP, Munzinger J, et al.** Chlamydia trachomatis as possible cause of peritonitis and perihepatitis in young women. Br Med J 1978; 1:1022–1024.
8. **Paavonen J, Saikku P, von Knorring J, Aho K, Wang SP.** Association of infection with Chlamydia trachomatis with Fitz-Hugh-Curtis syndrome. J Infect Dis 1981; 144:176.
9. **Wang SP, Eschenbach DA, Holmes KK, Wager G, Grayston JT.** Chlamydia trachomatis infection in Fitz-Hugh-Curtis syndrome. Am J Obstet Gynecol 1980; 138:1034–1038.
10. **Dalaker K, Gjonnaess H, Kvile G, Urnes A, Anestad G, Bergan T.** Chlamydia trachomatis as a cause of acute perihepatitis associated with pelvic inflammatory disease. Br J Vener Dis 1981; 57:41–43.
11. **Wolner-Hanssen P, Svensson L, Westrom L, Mardh PA.** Isolation of Chlamydia trachomatis from the liver capsule in Fitz-Hugh-Curtis syndrome. N Engl J Med 1982; 306:113.
12. **Semchyshyn S.** Fitz-Hugh and Curtis syndrome. J Reprod Med 1979; 22:45–48.
13. **Onsrud M.** Perihepatitis in pelvic inflammatory disease—association with intrauterine contraception. Acta Obstet Gynecol Scand 1980; 59:69–71.
14. **Pletcher JR, Slap GB.** Pelvic inflammatory disease. Pediatr Rev 1998; 19:363–367.
15. **Vickers F, Maloney P.** Gonococcal perihepatitis. Arch Intern Med 1964; 114:120–123.
16. **Holm-Nielsen P.** Right upper quadrant pain in salpingitis and other abdominal diseases explained by absorption of exudates from the peritoneal cavity through the diaphragm. Acta Chir Scand 1953; 104:435–446.
17. **Lopez-Zeno JA, Keith LG, Berger GS.** The Fitz-Hugh-Curtis syndrome revisited. Changing perspectives after half a century. J Reprod Med 1985; 30:567–582.
18. **Scott G.** Subdiaphragmatic gonorrheal abscess. JAMA 1931; 96:1681–1682.
19. **Banerjee B, Rennison A, Boyes BE.** Sonographic features in a case of Fitz-Hugh-Curtis syndrome masquerading as malignancy. Br J Radiol 1992; 65:342–344.
20. **Money DM, Hawes SE, Eschenbach DA, et al.** Antibodies to the chlamydial 60 kd heat-shock protein are associated with laparoscopically confirmed perihepatitis. Am J Obstet Gynecol 1997; 176:870–877.
21. **Wolner-Hanssen P.** Oral contraceptive use modifies the manifestations of pelvic inflammatory disease. Br J Obstet Gynaecol 1986; 93:619–624.
22. **Conway DJ, Holland MJ, Campbell AE, et al.** HLA class I and II polymorphisms and trachomatous scarring in a Chlamydia trachomatis-endemic population. J Infect Dis 1996; 174:643–646.
23. **Patton DL, Kuo CC, Wang SP, Halbert SA.** Distal tubal obstruction induced by repeated Chlamydia trachomatis salpingeal infections in pig-tailed macaques. J Infect Dis 1987; 155:1292–1299.
24. **Brunham RC, Peeling RW.** Chlamydia trachomatis antigens: role in immunity and pathogenesis. Infect Agents Dis 1994; 3:218–233.
25. **Stamm WE.** Chlamydia trachomatis infections: progress and problems. J Infect Dis 1999; 179:S380–383.
26. **Counselman FL.** An unusual presentation of Fitz-Hugh-Curtis syndrome. J Emerg Med 1994; 12:167–170.
27. **Jacobson L, Westrom L.** Objectivized diagnosis of acute pelvic inflammatory disease. Diagnostic and prognostic value of routine laparoscopy. Am J Obstet Gynecol 1969; 105:1088–1098.
28. **Katzman DK, Friedman IM, McDonald CA, Litt IF.** Chlamydia trachomatis Fitz-Hugh-Curtis syndrome without salpingitis in female adolescents. Am J Dis Child 1988; 142:996–998.
29. **Ris HW.** Perihepatitis (Fitz-Hugh-Curtis syndrome). A review and case presentation. J Adolesc Health Care 1984; 5:272–276.
30. **McCormack WM.** Pelvic inflammatory disease. N Engl J Med 1994; 330:115–119.
31. **Gatt D, Jantet G.** Perisplenitis and perinephritis in the Curtis-Fitz-Hugh syndrome. Br J Surg 1987; 74:110–112.
32. **Garcia Compean D, Blanc P, d'Abrigeon G, Larrey D, Michel H.** Fitz-Hugh and Curtis syndrome [in French]. Presse Med 1995; 24:1348–1351.
33. **Dinerman LM, Elfenbein DS, Cumming WA.** Clinical Fitz-Hugh-Curtis syndrome in an adolescent. Ultrasonographic findings. Clin Pediatr (Phila) 1990; 29:532–535.
34. **Romo LV, Clarke PD.** Fitz-Hugh-Curtis syndrome: pelvic inflammatory disease with an unusual CT presentation. J Comput Assist Tomogr 1992; 16:832–833.
35. **van Dongen PW.** Diagnosis of Fitz-Hugh-Curtis syndrome by ultrasound. Eur J Obstet Gynecol Reprod Biol 1993; 50:159–162.
36. **Schoenfeld A, Fisch B, Cohen M, Vardy M, Ovadia J.** Ultrasound findings in perihepatitis associated with pelvic inflammatory disease. J Clin Ultrasound 1992; 20:339–342.
37. **Tsubuku M, Hayashi S, Terahara A, Furukawa T, Ohmura G.** Fitz-Hugh-Curtis syndrome: linear contrast enhancement of the surface of the liver on CT. J Comput Assist Tomogr 2002; 26:456–458.
38. **Keane JA, McKimm RJ, David CM.** Perihepatitis associated with pelvic infection: the Fitz-Hugh-Curtis syndrome. NZ Med J 1982; 95:725–728.
39. **Miettinen AK, Heinonen PK, Laippala P, Paavonen J.** Test performance of erythrocyte sedimentation rate and C-reactive protein in assessing the severity of acute pelvic inflammatory disease. Am J Obstet Gynecol 1993; 169:1143–1149.
40. **McCormick M, DelCastillo J, Berk RS.** An atypical presentation of the Fitz-Hugh-Curtis syndrome. J Emerg Med 1990; 8:55–58.
41. **Clinical Effectiveness Group.** National guideline for the management of pelvic infection and perihepatitis. Sex Transm Infect 1999; 75:S54–56.
42. **Centers for Disease Control and Prevention.** Sexually transmitted diseases treatment guidelines 2002. MMWR Recomm Rep 2002; 51:1–78.
43. **McNeeley SG, Hendrix SL, Mazzoni MM, Kmak DC, Ransom SB.** Medically sound, cost-effective treatment for pelvic inflammatory disease and tuboovarian abscess. Am J Obstet Gynecol 1998; 178:1272–1278.
44. **Reichert JA, Valle RF.** Fitz-Hugh-Curtis syndrome. A laparoscopic approach. JAMA 1976; 236:266–268.

ADDRESS: *Nadja G. Peter, MD, Craig-Dalsimer Division of Adolescent Medicine, Children's Hospital of Philadelphia, 34th & Civic Center Boulevard, Philadelphia, PA 19104; e-mail peter@email.chop.edu.*