

# Clinical Communications: Adults



## ACUTE FITZ-HUGH-CURTIS SYNDROME IN A MAN DUE TO GONOCOCCAL INFECTION

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**Abstract—Background:** Fitz-Hugh-Curtis syndrome is a rare extra-pelvic complication of genital infection involving the perihepatic capsule. Most cases have been described in women in association with pelvic inflammatory disease; in rare cases it has been reported in men. Because the main symptom is acute abdominal pain, and laboratory and imaging findings are frequently nonspecific, the differential diagnosis, considering other gastrointestinal or renal diseases, can be difficult in the early stage of the syndrome, leading to frequent misdiagnosis and mismanagement. **Case Report:** We report a case of Fitz-Hugh-Curtis syndrome in a 26-year-old man who first presented to the emergency department with acute abdominal pain, vomiting, and fever. **Diagnosis** was possible on the basis of clinical signs of orchiepididymitis, abnormal ultrasound findings, and specialist consultation with the Sexually Transmitted Infection Clinic. An acute gonococcal infection was revealed, which was complicated by a collection of free perihepatic fluid and a subcapsular hypoechoic focal lesion. Prompt antibiotic therapy was established, with complete resolution of the symptoms within a few days. **Why should an emergency physician be aware of this?:** Awareness of the clinical presentation, imaging, and laboratory findings during the acute phase of Fitz-Hugh-Curtis syndrome could help emergency physicians to make an early diagnosis and to correctly manage such patients. Improved diagnostic skills could pre-

vent chronic complications that are especially a risk in the case of delayed or minor genitourinary symptoms. © 2015 Elsevier Inc.

**Keywords—**abdominal pain; Fitz-Hugh-Curtis syndrome; *Neisseria gonorrhoeae*; liver subcapsular fluid; genitourinary symptoms

### INTRODUCTION

Fitz-Hugh-Curtis syndrome (FHCS) is characterized by inflammation of the perihepatic capsule without involvement of liver parenchyma (1). It is thought to be primarily a consequence of *Chlamydia trachomatis* infection, although *Neisseria gonorrhoeae* has also been implicated. Most of the cases have been described in women affected by pelvic inflammatory disease (PID), but sporadic cases in men have been reported (2). The main symptom is acute abdominal pain in the right upper quadrant, alone or in association with diffuse pain in the lower abdomen, leading most patients to present to the emergency department (ED). Fever or urogenital symptoms are rarely present in FHCS. The differential diagnosis can be challenging, and this syndrome might be confused

with different gastrointestinal or renal diseases, such as cholecystitis, duodenal ulcer, acute appendicitis, or pyelonephritis (3,4). Acute FHCS can be diagnosed in the early stage through noninvasive methods, such as abdominal ultrasound or computed tomography (CT) scan, and readily treated with antibiotic therapy. Laparoscopy remains the main diagnostic and therapeutic tool in the chronic stage (5–7).

We report a case of acute FHCS in a man infected by gonorrhea, with abnormal liver ultrasound findings, who was promptly treated with antibiotic therapy.

### CASE REPORT

A 26-year-old man from Pakistan presented to the ED for acute abdominal pain, vomiting, and fever. These symptoms had been present for 5 days with progressive deterioration in the patient's clinical status. The abdominal pain was diffuse, but was mostly localized in the epigastrium. It was not possible to collect the medical history because of the language barrier. On examination, the abdomen was painful and diffusely tender, there was an absence of rebound tenderness. Temperature was 37.4°C. His urine sample taken on admission was positive for a high number of leukocytes (300/ $\mu$ L) but negative for the presence of bacteria. Chest and abdominal x-ray studies performed on hospital admission appeared normal and excluded intestinal perforation or occlusion. Thirty-six hours after admission, the patient was transferred to the Gastroenterology Unit for further investigations. At this time, he reported an intense burning sensation during urination and the clinical picture was slightly changed; on examination, the abdomen was soft and tender in the right upper quadrant and in the hypogastric region. There was no abdominal guarding or rigidity, and no Murphy sign or rebound tenderness. His vital signs were within normal limits, with persistence of mild fever (37.8°C). Laboratory workup showed a white blood cell (WBC) count of 27.130/mm<sup>3</sup> with 87% neutrophils, an elevated C-reactive protein (CRP) (20.33 mg/dL) and erythrocyte sedimentation rate (ESR) (70 mm), with normal liver and renal function tests. Abdominal ultrasound showed a small perihepatic fluid collection and a single subcapsular hypoechoic focal lesion. Forty-eight hours after hospital admission the patient complained of acute scrotal pain, and there was evidence of left orchiepididymitis. At this time, he acknowledged engaging in unsafe sexual intercourse in recent days. Testicular ultrasound was performed, and it showed signs of inflammation with increased vascularization on color Doppler. On consultation and workup at the Sexually Transmitted Infection (STI) Clinic, the microscopic evaluation of urethral smear was positive for diplococci and a high number of neutrophils (>5 WBC/high-power field). A real-time

polymerase chain reaction (PCR) for simultaneous detection of *C. trachomatis* and *N. gonorrhoeae* (Siemens Healthcare Diagnostic, Tarrytown, NY) was performed on the patient's urine specimen, with a positive result for DNA amplification of copy gene locus of *N. gonorrhoeae* *pivNG* gene. Antibiotic therapy was established with ceftriaxone 500 mg intramuscularly and azithromycin 1 g orally. After treatment, the patient reported complete resolution of his symptoms and all laboratory parameters normalized within a few days. After 7 days of hospitalization, the patient was discharged. An abdominal ultrasound and medical consultation at the STI Clinic were planned after 3 weeks. The patient did not attend the medical appointment, therefore, no information about follow-up is available.

### DISCUSSION

FHCS is characterized by inflammation of the perihepatic capsule without involvement of liver parenchyma, resulting in adhesion of the anterior hepatic face and abdominal wall. It was first described in the 1930s as a consequence of pelvic inflammation due to gonococcal infection (8,9). Successively, in 1985, *C. trachomatis* was recognized as the most frequent pathogen isolated in cases of FHCS (10). However, cases secondary to *N. gonorrhoeae* infection are still present and rare cases after genital tuberculosis have been described recently (11,12).

The typical presentation occurs in sexually active young women in association with PID, and only a few cases have been reported in men (2). Bacterial spread through the peritoneal cavity from infected fallopian tubes has been suggested as the main mechanism of development of FHCS. In male patients, hematogenous and lymphatic spread to the liver has been hypothesized (2,4). The typical symptom is right upper quadrant abdominal pain, alone or in association with lower abdominal pain. Different localizations of pain, mainly in the epigastrium, left upper quadrant, or lower abdomen alone have also been described. Fever or genitourinary symptoms are rarely present. This clinical presentation leads to frequent misdiagnosis as different gastrointestinal and renal diseases. Acute FHCS is characterized by very nonspecific symptoms, mimicking cholecystitis, biliary colic, appendicitis, liver abscess, duodenal ulcer, pyelonephritis and, less frequently, hepatitis, pleurisy, and herpes zoster infection (3,4). Laboratory findings are usually nonspecific, with elevated WBC, CRP, and ESR. Liver and renal function tests are often normal, helping in differentiating from hepatobiliary or renal diseases. Microbiological analysis for *C. trachomatis* and *N. gonorrhoeae* is highly advisable when the diagnosis of FHCS is suspected, because these pathogens are implicated in most of the cases. The etiological role of

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