J. Phys. Ther. Sci.
27: 1641–1644, 2015

Original Article

Fitz-Hugh-Curtis syndrome: clinical diagnostic value of dynamic enhanced MSCT

Pei-Yuan Wang[1–3], Lin Zhang[1, 2], Xia Wang[3], Xin-Jiang Liu[2], Liang Chen[2], Xu Wang[2], Bin Wang[3]*

[1] *Shandong Medical Imaging Research Institute, Shandong University, China*
[2] *Department of Radiology, Affiliated Hospital of Binzhou Medical University, China*
[3] *Medical Imaging Research Institute, Binzhou Medical University: No. 346 Guanhai Road, Laishan District, Yantai 264003, China*

Abstract. [Purpose] This study aimed to investigate the clinical diagnostic value of dynamic enhanced multislice computed tomography (MSCT) for Fitz-Hugh-Curtis syndrome (FHCS). [Subjects and Methods] This study retrospectively analyzed the clinical features and manifestations of scanning and dynamic enhanced MSCT in 19 patients with FHCS. [Results] MSCT scans showed different degrees of liver capsule thickness in the lesion area: seven cases of sub-capsular effusion and three cases with a small amount of pleural effusion; thickness of the liver capsular arterial phase showing significant enhancement in 17 cases, and slight enhancement in two; portal venous and delayed phase enhancement decreased with no clear boundary of the liver parenchyma; and adjacent hepatic parenchymal involvement in five cases, in which the arterial phase appeared to have patchy or triangular enhancement, and unclear portal vein and delayed phase imaging findings. MSCT revealed pelvic inflammatory disease in 14 cases, peritonitis in two, endometritis combined with bilateral ovarian abscesses in two, and a tube-ovarian abscess in one. [Conclusion] Dynamic enhanced MSCT can accurately display liver capsule lesions and possible pelvic inflammatory diseases related to FHCS, suggest the infection source, and have high application value for making early, accurate diagnoses and improved prognosis.
Key words: Tomography, X-ray compute, Perihepatitis

(This article was submitted Dec. 10, 2014, and was accepted Feb. 7, 2015)

INTRODUCTION

Perihepatitis (Fitz-Hugh-Curtis syndrome [FHCS]) is a syndrome in which a pelvic infection spreads to the liver's diaphragmatic surface, causes liver capsular inflammation without involvement of the hepatic parenchyma, and is accompanied by right upper quadrant pain, which was reported by Curtis[1] and Fitz-Hugh[2] in 1930 and 1934, respectively. The acute-stage clinical manifestations of this disease, including sudden right upper quadrant pain, muscular tension, and pain may become aggravated with changes in breathing and posture but have no obvious characteristics, so it is often misdiagnosed as cholecystitis, pleurisy, or pyelonephritis[3–5]. In the past, the definitive diagnosis of FHCS mainly required laparoscopy or exploratory laparotomy, the typical finding being the detection of violin string-like adhesions, and the causative organisms being identified in lesion specimens of the hepatic capsule.

With the development and availability of multislice computed tomography (MSCT), CT, a noninvasive diagnostic procedure, has become the main diagnostic method for FHCS. Abdominal and pelvic MSCT examinations, especially dynamic enhancement examinations as a noninvasive method, combined with laboratory and etiological examination, has important value in the diagnosis of this disease[6]. Here, we retrospectively analyzed 19 cases of FHCS that were definitely diagnosed by using MSCT and laboratory examination to determine the value of dynamic enhanced MSCT in the diagnosis of FHCS, increase our understanding of this disease, and improve diagnostic accuracy.

SUBJECTS AND METHODS

From January 2008 to June 2013, 19 patients undergoing treatment for FHCS in the Affiliated Hospital of Binzhou Medical College were enrolled and their complete clinical and imaging data were collected. All patients were females and aged 20–38 years (mean, 28.7 years). During hospitalization, all patients underwent blood, serum, and vaginal pathogen examinations. This study was conducted in accordance with the declaration of Helsinki, and with approval from the Ethics Committee of Binzhou Medical University. Written informed consent was obtained from all participants.

Abdominal and pelvic MSCT scans were performed using an American GE 64 layer Lightspeed VCT (GE Lightspeed VCT, USA). The scan started at the diaphragm and

*Corresponding author. Bin Wang (E-mail: binwangdoc@126.com)

ended at the pubic symphysis. Before enhanced scanning was performed, the patients underwent an abdominal and pelvic CT scan. During enhanced scanning, 100 mL of non-ionic contrast agent (Omnipaque 300 mgI; GE Healthcare, Shanghai, China) was injected with a high-pressure injector via the medial cubital vein with a flow rate of 3.0–3.5 mL/s; arterial, portal venous, and delayed phase scans were performed 28, 60, and 180 s after injection of the contrast agent. Scan parameters were as follows: 120 kV; 300 mA; matrix, 512 × 512, collimation, 0.625 mm; reconstruction thickness, 5 mm; pitch, 0.984; and a large scanning field. The original data were transmitted to the GE AW 4.3 workstation for post-processing to obtain multiplanar volume reconstruction images. The original image combined with the reconstructed image was used to observe disease manifestations and characteristics.

RESULTS

All of the patients visited our hospital for pain in the right upper abdominal quadrant on the first visit, with lower abdominal pain in six patients (31.6%) and right-sided pain in two (10.5%). Physical examination revealed right upper quadrant abdominal muscle tension in seven patients and abdominal muscle tension in two patients. Laboratory examinations revealed increased total white blood cells and neutrophils (68.4%) in 13 patients, erythrocyte sedimentation rate (ESR) increased in 11 (22–48 mm/h; normal, 0–20 mm/h), elevated C-reactive protein (CRP; 16–43 mg/L; normal, 0–8.00 mg/L) in 16; polymerase chain reaction detection of vaginal secretions showed *Chlamydia trachomatis* in 14 patients (73.7%) and *Neisseria gonorrhoeae* in two (10.5%). Clinical diagnoses included 14 cases of acute pelvic inflammatory disease (PID), two cases of acute peritonitis, two cases of endometriosis and ovarian cyst, and one case of a tubal-ovarian abscess.

An MSCT scan of the liver capsule showed different degrees of thickening, 13 cases of linear or zonal homogeneous thickness, and six cases of inhomogeneous thickness with slightly increased density within the background of fatty liver (Fig. 1A); seven cases of hepatic subcapsular effusion, including five cases of extensive effusion and two cases of localized effusion; and three cases complicated by a small right pleural effusion. None of the cases was complicated by a hepatic peripheral organ disease.

Dynamic enhanced MSCT scanning of the arterial phase in the liver capsule showed different degrees of thickening, in which significant enhancement was seen in 17 cases and slight enhancement in two; in the portal venous phase and delayed phase, the degree of enhancement of liver capsule thickening decreased compared to that in the arterial phase, which was similar to the normal liver parenchyma (Fig. 1B–1D); and five cases of adjacent hepatic parenchymal involvement showed patchy or triangular enhancement with no clear boundary with a thickened liver capsule, while the portal venous and delayed phases were not clearly visible (Fig. 2). An enhanced MSCT scan revealed pelvic chip heterogeneous enhancement with different amounts of pelvic effusion as well as pelvic fat diffuse infiltration in 14 cases, which was suggestive of PID (Fig. 3), including one case of bilateral ovarian abscess, two cases of peritonitis, two cases of endometritis and bilateral ovarian abscess (Fig. 4), and one case of ovarian fallopian tube abscess. No exudative lesions were visible in the right paracolic gutter area in any of the cases (Figs. 3B and 4B).

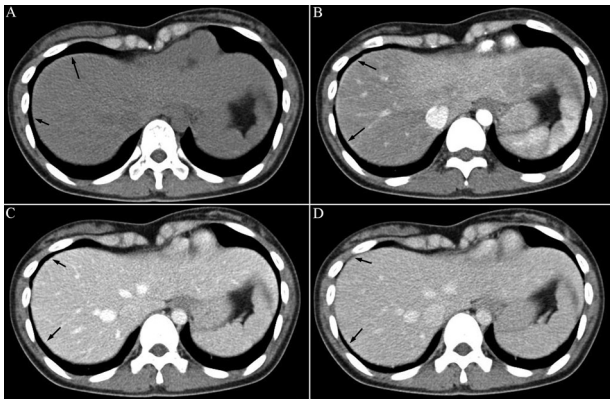

Fig. 1. A 27-year-old married woman with right upper abdominal pain for 3 days. A. Plain scan: visible uniform thickening of the liver capsule within a background of fatty liver, with slightly higher intensity than the liver parenchyma; B. Arterial phase: thickened liver capsule is obviously enhanced; C. Portal vein phase: the degree of liver capsule enhancement has decreased; D. Delay period phase: liver capsule is enhanced similarly to liver parenchyma, with no clear boundary.

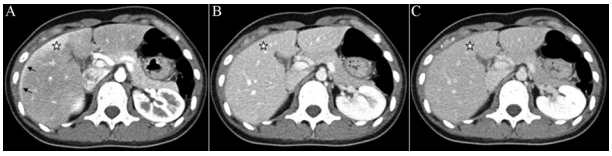

Fig. 2. A 23-year-old unmarried woman with right upper quadrant abdominal pain for 7 days was diagnosed with acute pelvic inflammatory disease. A. Arterial phase: the liver capsule is a homogeneously thickened strip, with obvious enhancement (arrow), and shows a sub-capsular parenchymal triangle abnormally enhanced zone (asterisk); B. Portal venous phase, C. Delayed phase, liver capsule and the involved hepatic parenchyma enhancement not displayed clearly.

DISCUSSION

FHCS is always secondary to PID, with an incidence rate of 4–27%[7]. In 1930 and 1934, Curtis[1] and Fitz-Hugh[2] successively reported FHCS, and determined that *N. gonorrhoeae* is the major causative pathogen. In 1978, Muller-Schoop et al.[8] reported that *C. trachomatis* was the new causative pathogen, while recent studies have demonstrated that *C. trachomatis* is the much more common pathogen implicated in FHCS[9–11]. You et al.[12] and Woo et al.[13] reported that *C. trachomatis* pathogens accounted for 89% and 86% of FHCS cases, respectively. In this study, *C. trachomatis*

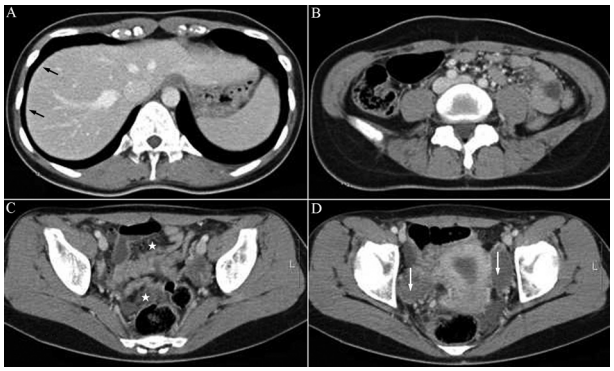

Fig. 3. A 27-year-old woman with right upper abdominal pain and pelvic pain for 4 days was diagnosed with pelvic inflammatory disease. A–D = portal venous phase. A. Showing a homogeneously thickened strip of enhancement (black arrow); B. Showing lesion in the right paracolic gutter area no exact anomaly density; C. Showing pelvic fat spaces with increased density, inhomogeneous enhancement, and visible rectal effusion (cross); D. Showing bilateral ovarian abscess (white arrow) and surrounding uterus effusion.

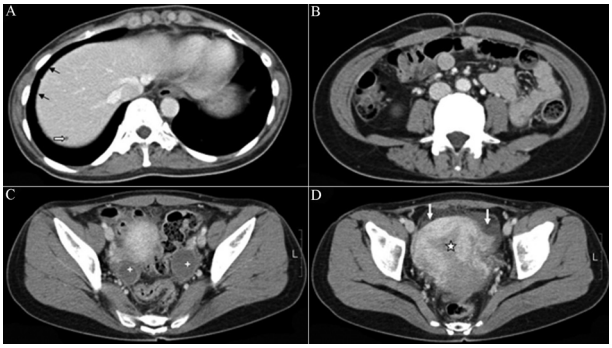

Fig. 4. A 21-year-old woman with right upper abdominal pain and pelvic pain for 4 days was diagnosed with endometritis and bilateral ovarian abscess. A–D = portal venous phase. A. Showing homogeneously thickened strip of enhancement (black arrow) and liver cysts (hollow arrow); B. Showing lesion in the right paracolic gutter area with no exact anomaly density; C. Showing bilateral ovarian abscess (cross); D. Showing endometrial thickening, low degree of enhancement (asterisk), and surrounding uterus effusion (white arrow).

accounted for 73.7% of causative pathogens (14/19). It has also been reported that genital tuberculosis, appendicitis, and laparoscopic operation are associated with FHCS[14, 15]. However, the route of infection and the pathogenesis of FHCS remain unclear, and the possible mechanisms include pelvic inflammatory effusion spreading along the right paracolic gutter directly to the diaphragm, hematogenous dissemination, and lymphatic spread to cause inflammation and adhesion of the liver capsule[12]. No patient in this study had definite signs of direct spread.

The main clinical manifestations of FHCS are right upper quadrant pain that worsens during deep breathing, coughing, or postural changes with fixed tenderness; when it is accompanied by PID, it may manifest as lower abdominal pain or generalized abdominal pain with no obvious characteristics, and so is easily misdiagnosed as acute cholecystitis, gallstones, pleural membrane inflammation, or acute pyelonephritis, et al.[3–5, 12, 16–18] during the first visit. Patients may also have fever, increased vaginal secretions, and mechanical intestinal obstruction.

The results of laboratory examinations have revealed that some patients had an elevated white cell count, increased ESR, and elevated CRP, while most had normal liver function. Through antibiotic treatment, most patients can be cured[19]. In this study, 68.4% of patients had elevated white blood cell counts, 57.9% had elevated ESR (up to 48 mm/h), while 84.2% had an elevated CRP, findings that were consistent with the results of You et al[12].

Early diagnosis is critical for the clinical treatment and prognosis of FHCS. In the past, the diagnosis of FHCS mainly depended on laparoscopy or laparotomy, and laparoscopy is still considered the gold standard in the diagnosis of FHCS[20]. With the development of MSCT in clinical applications, because of its large scanning range, speed, volume scanning, and clear images, many advantages such as post-processing functionality and non-invasive nature, it has gradually replaced laparoscopy to become the main diagnostic technology for FHCS.

CT manifestations are closely related to pathological FHCS staging. Acute fibrinous inflammation of the liver capsule surface was seen in the acute phase of FHCS, and MSCT examinations often show no obvious abnormalities. The FHCS pathological changes of the chronic phase were liver capsule fibrosis, hyaline degeneration, localized or widespread liver capsule thickening, and band adhesions between the liver surface and the abdominal wall[10, 16, 21, 22]. MSCT scanning can display homogeneous or inhomogeneous thickening of the liver capsule with or without hepatic subcapsular effusion or pleural effusion, but it cannot display the disease extent or whether local fiber adhesions exist and to what degree.

MSCT dynamic enhancement in the arterial phase can better show enhanced thickening of the liver capsule and whether the adjacent liver tissue is involved, which has important value in the diagnosis of FHCS. Wang et al.[16] classified liver capsule thickening shape and range on CT and magnetic resonance imaging (MRI) examinations of 21 patients with FHCS. Kim et al.[23] thought that dynamic enhanced MSCT is of benefit for observing the site, shape, and thickness changes of liver capsule enhancement in the arterial phase, while the characteristics of liver capsule enhancement of the portal and delayed phases can reflect the early changes in fibrosis. In this study, all patients had different degrees of liver capsule thickening: 17 had significant enhancement and two had slight enhancement in the arterial phase, while five had liver parenchyma involvement, showing patchy or triangular enhancement with no clear boundaries, which proved that MSCT dynamic enhancement has significance in the diagnosis of FHCS. Hepatic subcapsular effusion was mostly diffuse (7/9) and less localized.

In this study, the two cases of localized effusion may have

been due to localized adhesions caused by chronic inflammation, but this must be further demonstrated by laparoscopy. Small amounts of subcapsular effusion are better visualized on MRI T2WI than MSCT. After systemic standardized antibiotic treatment, liver capsule enhancement signs of FHCS can completely disappear. Abdominal and pelvic MSCT examination can enable observation of the changes in the pelvic genital organs and fatty space at the same time, and if the PID sign is found, it helps in the definite diagnosis of FHCS. Furthermore, dynamic enhanced MSCT can be used to detect the lesions in the adjacent organs or peritoneum and display information such as site, shape, and enhancement characteristics of liver capsular thickening induced by the liver lesions, which is of great value for the differential diagnosis of liver capsular thickening caused by FHCS as well as gallbladder or pancreatic lesions or peritoneal cancer. In addition, dynamic enhancement of MSCT combined with ultrasonic inspection technology can differentiate subcapsular abnormal perfusion caused by FHCS, fatty liver, and superior vena cava obstruction[12, 24].

There is currently a lack of appropriate standards and references for the clinical and imaging staging of FHCS. This study did not focus on FHCS staging or image staging manifestations. Hong et al.[25] thought that the diagnosis of FHCS should focus on perihepatitis rather than etiology and symptoms. Therefore, in the diagnosis of FHCS, the advantage of using dynamic enhanced MSCT to identify liver capsule lesions will be more widely applicable in the diagnosis of suspected cases.

In conclusion, in a woman of reproductive age who presents with acute right upper quadrant pain with or without lower abdominal pain, the possibility of FHCS should be kept in mind. A dynamic enhanced MSCT scan can show obvious enhancement and thickening of the liver capsule in the arterial phase and accurately visualize the associated PID, which is important for the early diagnosis of FHCS. In addition to the early diagnosis of FHCS, pathogenesis, standards, and references for clinical and imaging staging require future investigation and policy making.

REFERENCES

1) Curtis AH: A case of adhesionin the right upper quadrant. J Am Med Assoc, 1930, 94: 1221–1222. [CrossRef]
2) Fitz-Hugh T: Acute gonococcic peritonitis of the right upper quadrant in women. J Am Med Assoc, 1934, 102: 2094–2096. [CrossRef]
3) McCormick M, DelCastillo J, Berk RS: An atypical presentation of the Fitz-Hugh-Curtis syndrome. J Emerg Med, 1990, 8: 55–58. [Medline] [CrossRef]
4) Di Rocco G, Giannotti D, Collalti M, et al.: Acute abdominal pain in a 24-year-old woman: Fitz-Hugh-Curtis syndrome associated with pyelonephritis. Clinics (Sao Paulo), 2012, 67: 1493–1495. [Medline] [CrossRef]
5) Lee SC, Nah BG, Kim HS, et al.: [Two cases of Fitz-Hugh-Curtis syndrome in acute phase]. Korean J Gastroenterol, 2005, 45: 137–142. [Medline]
6) Chung HJ, Choi HY, Cho YJ, et al.: [Ten cases of Fitz-Hugh-Curtis syndrome]. Korean J Gastroenterol, 2007, 50: 328–333. [Medline]
7) Peter NG, Clark LR, Jaeger JR: Fitz-Hugh-Curtis syndrome: a diagnosis to consider in women with right upper quadrant pain. Cleve Clin J Med, 2004, 71: 233–239. [Medline] [CrossRef]
8) Müller-Schoop JW, Wang SP, Munzinger J, et al.: Chlamydia trachomatis as possible cause of peritonitis and perihepatitis in young women. BMJ, 1978, 1: 1022–1024. [Medline] [CrossRef]
9) Huang HH, Tsai CM, Tyan YS: Unusual cause should be kept in mind of abdominal pain in female patient. Fitz-Hugh-Curtis syndrome. Gastroenterology, 2011, 140: e7–e8. [Medline] [CrossRef]
10) Le Moigne F, Lamboley JL, Vitry T, et al.: [Usefulness of contrast-enhanced CT scan for diagnosis of Fitz-Hugh-Curtis syndrome]. Gastroenterol Clin Biol, 2009, 33: 1176–1178. [Medline] [CrossRef]
11) Antonie F, Billiou C, Vic P: [Chlamydia trachomatis Fitz-Hugh-Curtis syndrome in a female adolescent]. Arch Pediatr, 2013, 20: 289–291. [Medline] [CrossRef]
12) You JS, Kim MJ, Chung HS, et al.: Clinical features of Fitz-Hugh-Curtis Syndrome in the emergency department. Yonsei Med J, 2012, 53: 753–758. [Medline] [CrossRef]
13) Woo SY, Kim JI, Cheung DY, et al.: Clinical outcome of Fitz-Hugh-Curtis syndrome mimicking acute biliary disease. World J Gastroenterol, 2008, 14: 6975–6980. [Medline] [CrossRef]
14) Sharma JB, Roy KK, Gupta N, et al.: High prevalence of Fitz-Hugh-Curtis Syndrome in genital tuberculosis. Int J Gynaecol Obstet, 2007, 99: 62–63. [Medline] [CrossRef]
15) Kazama I, Nakajima T: A case of fitz-hugh-curtis syndrome complicated by appendicitis conservatively treated with antibiotics. Clin Med Insights Case Rep, 2013, 6: 35–40. [Medline] [CrossRef]
16) Wang CL, Guo XJ, Yuan ZD, et al.: Radiologic diagnosis of Fitz-Hugh-Curtis syndrome. Chin Med J (Engl), 2009, 122: 741–744. [Medline]
17) Cho SH, Kim KH, Baek IH, et al.: Comparison of contraction rates of abdominal muscles of chronic low back pain patients in different postures. J Phys Ther Sci, 2013, 25: 907–909. [Medline] [CrossRef]
18) Noh KH, Kim JW, Kim GM, et al.: The influence of dual pressure biofeedback units on pelvic rotation and abdominal muscle activity during the active straight leg raise in women with chronic lower back pain. J Phys Ther Sci, 2014, 26: 717–719. [Medline] [CrossRef]
19) Yang HW, Jung SH, Han HY, et al.: [Clinical feature of Fitz-Hugh-Curtis syndrome: analysis of 25 cases]. Korean J Hepatol, 2008, 14: 178–184. [Medline] [CrossRef]
20) Cho JH, Namgung JS, Lee J, et al.: Analysis of biochemical markers related to Fatty liver patients. J Phys Ther Sci, 2014, 26: 1865–1868. [Medline] [CrossRef]
21) Demelo-Rodríguez P, González-Munera A, Tejerina F, et al.: Fitz-Hugh-Curtis syndrome in subacute/acute phase. Rev Clin Esp, 2013, 213: e49. [CrossRef]
22) Joo SH, Kim MJ, Lim JS, et al.: CT diagnosis of Fitz-Hugh and Curtis syndrome: value of the arterial phase scan. Korean J Radiol, 2007, 8: 40–47. [Medline] [CrossRef]
23) Kim JY, Kim Y, Jeong WK, et al.: Perihepatitis with pelvic inflammatory disease (PID) on MDCT: characteristic findings and relevance to PID. Abdom Imaging, 2009, 34: 737–742. [Medline] [CrossRef]
24) Cho HJ, Kim HK, Suh JH, et al.: Fitz-Hugh-Curtis syndrome: CT findings of three cases. Emerg Radiol, 2008, 15: 43–46. [Medline] [CrossRef]
25) Hong DG, Choi MH, Chong GO, et al.: Fitz-Hugh-Curtis Syndrome: single centre experiences. J Obstet Gynaecol, 2010, 30: 277–280. [Medline] [CrossRef]