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Case Report

Hepatic pseudolesion as an unusual presentation of Fitz-Hugh-Curtis syndrome ☆

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ABSTRACT

Fitz-Hugh-Curtis Syndrome is a rare disorder manifesting as a complication associated with Pelvic Inflammatory Disease. The initial presentation generally consists of concomitant right upper quadrant and lower abdominal pain. This syndrome is characterized by inflammation of the peritoneum with the involvement of hepatic capsule and the tissues surrounding the liver. Intrahepatic involvement is rare and not yet well investigated. An accurate interpretation of Computed Tomography and Magnetic Resonance Imaging findings is missing in the literature. This report presents a case of Fitz-Hugh-Curtis Syndrome in which Computed Tomography and Magnetic Resonance Imaging showed a region of heterogeneously decreased enhancement and abnormal signal intensity within the liver mimicking a lesion.

Introduction

Fitz-Hugh-Curtis Syndrome (FHCS), first described by Curtis and Fitz-Hugh in the 1930s, is a rare disorder manifesting as a complication associated with Pelvic Inflammatory Disease (PID) [1,2]. This syndrome is characterized by inflammation of the peritoneum with the involvement of the tissues surrounding the liver and the hepatic capsule, mainly as a consequence of ascending peritoneal spread and/or lymphohematogenous dissemination of pelvic infections to the perihepatic region [2,3]. Typically, the liver parenchyma is not affected during FHCS, and accurate descriptions of imaging findings concerning intrahepatic alterations are missing in the literature. We hereby describe an unusual case of Fitz-Hugh-Curtis Syndrome showing typical findings on abdominal Computed Tomography (CT) imaging, yet associated with the presence of a hepatic pseudolesion seen as a region of low attenuation relative to the rest of the liver and appearing as an area of abnormal signal intensity on Magnetic Resonance Imaging (MRI).

☆ Competing Interests: The authors have no conflicts of interest do declare.

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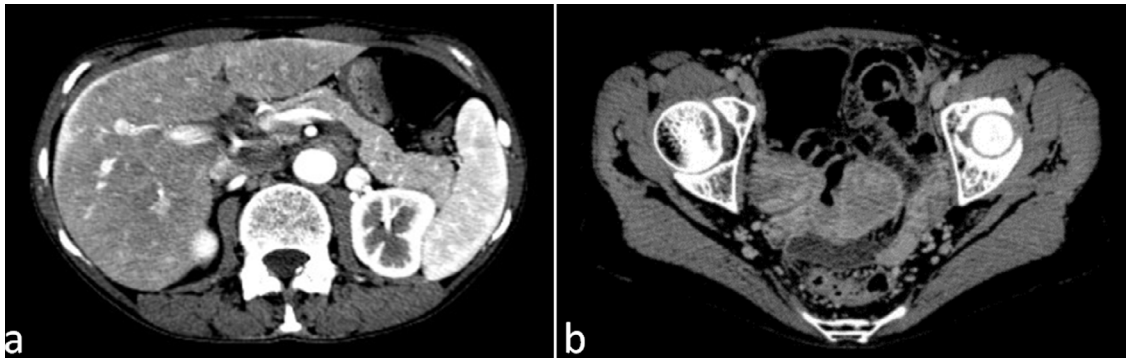

Fig. 1 – Contrast-enhanced abdominal CT on admission. (A) Axial contrast-enhanced CT scan in arterial phase shows thickening of the liver capsule associated with conspicuous increased perihepatic enhancement, suggesting perihepatitis compatible with Fitz-Hugh-Curtis Syndrome. (B) Axial contrast-enhanced CT image obtained at the level of the pelvis during portal-venous phase demonstrates free fluid within the pouch of Douglas with thickening and enhancement along the peritoneum and the peritoneal coverings of the uterus, suggesting the presence of an infectious-inflammatory process. Moreover, the right adnexal region appears swollen and inhomogeneous with surrounding inflammatory stranding, fallopian tube is more conspicuous due to wall thickening and enhancement, and uterine border is slightly indistinct: all common CT imaging features of PID.

Case Report

A 54year-old women presented to our department as outpatient to perform a pelvic ultrasound examination complaining of diffuse abdominal pain, mainly located in the right upper quadrant. She reported no other symptoms, and no apparent clinical signs were present. Her medical history and familial history were unremarkable. Laboratory investigations did not reveal any abnormalities: full blood count results, renal and liver function tests were all normal.

Pelvic ultrasonography revealed the presence of free intraperitoneal fluid within the pouch of Douglas. Such a non-specific finding and the persistence of abdominal pain prompted radiologists to explore further, and hence contrast-enhanced CT imaging of the abdomen and pelvis was performed.

Findings of contrast-enhanced CT through upper abdomen during arterial phase demonstrated thickening of the liver capsule associated with conspicuous increased perihepatic enhancement and fat stranding adjacent to the liver edge, suggesting perihepatitis. Images obtained at the level of the pelvis showed diffuse swelling of the right adnexal region, with fallopian tube appearing more conspicuous due to wall thickening and enhancement, and uterus showing slightly indistinct borders. Free fluid within the pouch of Douglas and peritoneal thickening were also present: all common CT imaging features of PID (Fig. 1). At the same time, CT scan also revealed a large hypoattenuating lesion on arterial phase located within the liver. On delayed imaging, this area became isoattenuating relative to adjacent normal liver parenchyma, thus suggesting a region of heterogeneously decreased enhancement related to a perfusion defect. Indeed, portal-venous images revealed the presence of hypoattenuating linear areas along segmental branches of left portal vein in the exact same location where the lesion was observed, suspicious for thrombosis (Fig. 2).

Liver MRI with hepatobiliary-specific contrast agent was performed three days after CT and showed an ill-defined area of increased parenchymal signal intensity on T2-weighted images around the portal system at the same level, compatible with inflammatory changes (Fig. 3). Moreover, peri-hepatic and peri-splenic free fluid was also observed. MRI scans after contrast agent injection confirmed findings from CT imaging, and no alterations in the hepatobiliary phase were observed.

Despite the unexpected involvement of liver parenchyma, a diagnosis of Fitz-Hugh-Curtis Syndrome was made based on the persistent abdominal pain and imaging findings consistent with pelvic inflammatory disease associated with perihepatitis.

The patient was placed on oral antibiotic therapy with doxycycline 100 mg twice a day for 14 days, in combination with analgesic therapy. She returned to our department at the end of the treatment in good clinical condition and contrast-enhanced MRI of the liver was performed, showing complete disappearance of the parenchymal alteration of the liver on T2-weighted and contrast-enhanced images, as well as resolution of free intraperitoneal fluid (Fig. 4).

Discussion

Fitz-Hugh-Curtis Syndrome (FHCS) is characterized by perihepatic inflammation that typically occurs as a complication of Pelvic Inflammatory Disease (PID), mainly through ascending peritoneal spread and/or lympho-hematogenous dissemination of pathogenic organisms to the perihepatic region.

Due to its association with PID, FHCS typically affects childbearing women, with *Chlamydia Trachomatis* and *Neisseria Gonorrhoeae* representing the most common causative organisms. Clinically, FHCS presents with concomitant right upper quadrant and lower abdominal pain and it is often challenging for physicians to diagnose since it may mimic several

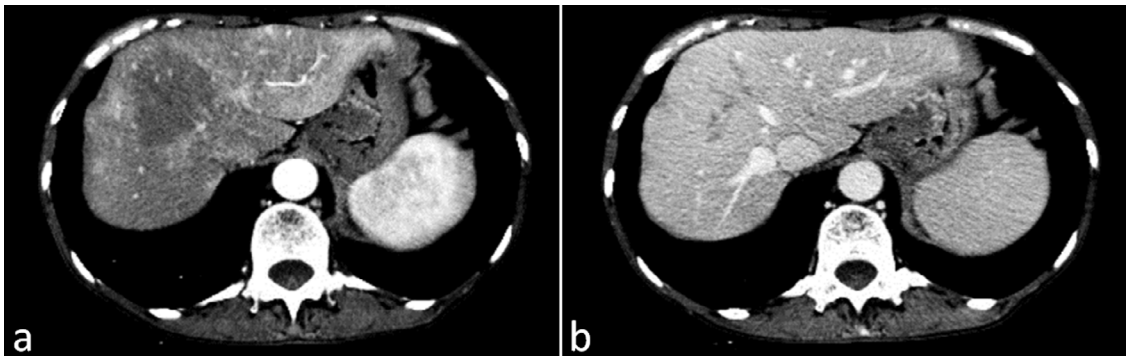

Fig. 2 – Contrast-enhanced abdominal CT on admission through the liver dome. (A) Axial contrast-enhanced CT image obtained at the level of the liver dome during arterial phase shows a large lesion seen as a region of heterogeneously decreased enhancement relative to the rest of the liver. (B) Axial contrast-enhanced CT image obtained during portal-venous phase reveals the presence of hypoattenuating linear areas along segmental branches of left portal vein in the exact same location where the lesion was observed, suspicious for thrombosis.

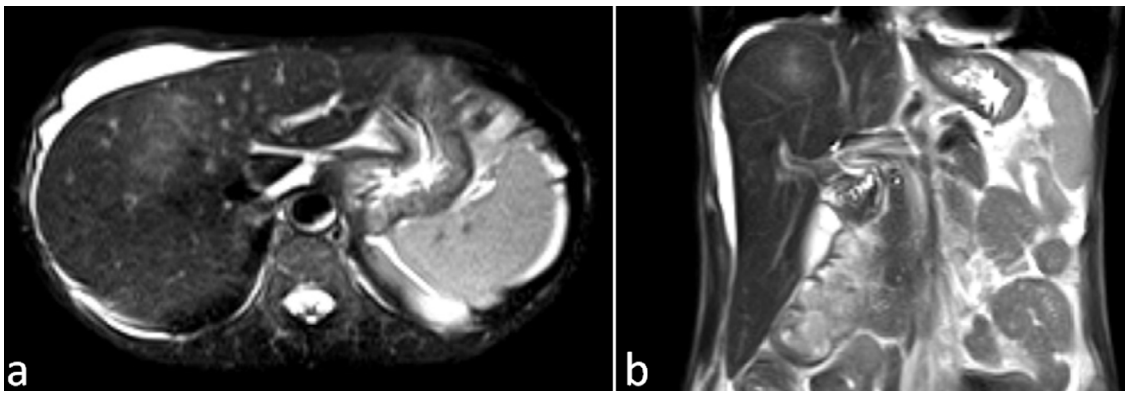

Fig. 3 – MRI of the liver performed three days after CT. (A) Axial T2-weighted sequence with fat suppression demonstrates an ill-defined area of increased parenchymal signal intensity around the portal system in the same position where the liver lesion was observed on CT, compatible with inflammatory changes. Peri-hepatic and peri-splenic free fluid is also present. (B) Coronal T2-weighted sequence with fat suppression confirming the findings.

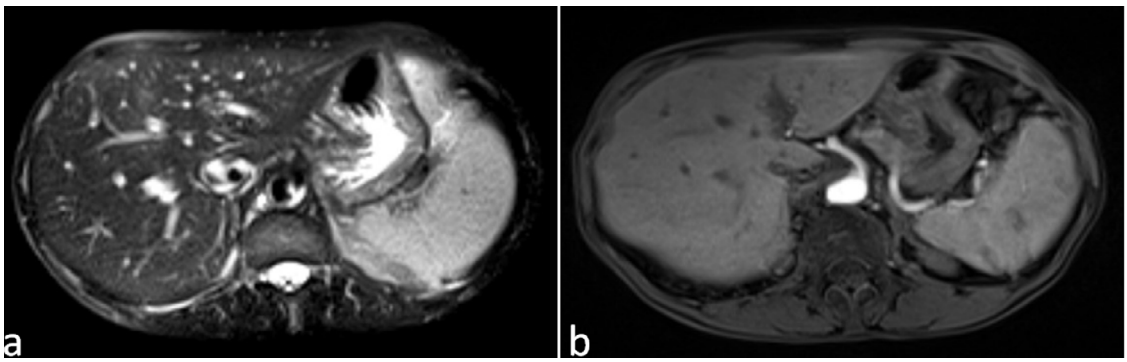

Fig. 4 – Contrast-enhanced MRI of the liver after completion of therapy. (A) Axial T2-weighted sequence with fat suppression shows disappearance of the parenchymal alteration of the liver and free intraperitoneal fluid. (B) Axial plane post-contrast T1-weighted image through the liver dome shows how the hepatic parenchymal alteration completely resolved on follow-up MRI after doxycycline treatment.

other pathologies, including gastrointestinal, hepatobiliary, and genitourinary diseases [4].

Physical examination findings may reveal abdominal tenderness, or a silent abdomen, while the presence of cervical motion tenderness and vaginal discharge could be helpful signs in suspected cases of PID. Laboratory tests may indicate inflammation with leucocytosis and elevated CRP, even though only up to 50% of women with PID have clinically significant leucocytosis [5].

Visualization of violin-string like adhesions with laparoscopy is the gold standard for diagnosing FHCS [6]. However, given the invasive nature of this procedure, a presumptive diagnosis is often made and diagnostic imaging techniques such as ultrasonography and computed tomography (CT) represent important tools in ruling out other differentials. Ultrasound imaging plays a part in assessing tubo-ovarian anomalies and other signs of PID in the pelvis, but has several limitations in confirming FHCS [7]. On the other hand, several studies have suggested that FHCS could be diagnosed radiologically, demonstrating sufficient data for the diagnosis of the syndrome by means of contrast-enhanced CT [8,9]. Cross-sectional imaging findings obtained with contrast-enhanced CT include thickening and increased enhancement along the hepatic capsule, with mild fat stranding adjacent to the liver, whereas involvement of the liver parenchyma typically does not occur during FHCS. Although its suspicious appearance, the anomalous large hypoattenuating area identified in our patient could be attributed to a localised mismatch in hepatic arterial versus portal venous blood supply. Indeed, linear areas suspicious for thrombosed segmental portal vein branches were observed during portal-venous phase to account for the abnormality. MRI images obtained three days after CT revealed increased T2 parenchymal signal intensity at the same level, suggesting inflammatory changes. In conjunction with findings of perihepatitis, the hepatic inflammatory reaction could have contributed to structural changes in liver sinusoidal endothelial cells, leading to thrombotic phenomena in portal and sinusoidal vessels. Follow-up MRI examination after antibiotic treatment showed complete resolution of the parenchymal alteration.

In summary, we described an unusual case of hepatic involvement, characterized by a suspicious region of heterogeneously decreased enhancement associated with parenchymal inflammatory changes, in a patient with Fitz-Hugh-Curtis Syndrome.

An imaging diagnosis of FHCS should be considered in any woman with clinical findings suggestive of PID, even in the presence of alterations involving the hepatic parenchyma.

FHCS is a benign entity with an optimal prognosis and its management involves conservative treatment using antibiotics like doxycycline and azithromycin, even though surgical evaluation might be considered in cases unresponsive to antibiotic therapy [5,6].

Knowledge of FHCS can prevent incorrect diagnosis and avoid invasive examinations. Clinical features of PID in association with typical CT imaging should raise concern for FHCS. The use of non-invasive diagnostic procedures is desirable, considering that FHCS is a benign condition that can be successfully treated administering appropriate antibiotics.

Patient consent

Written, informed consent was obtained from all patients for being included in the study for publication of their case.

REFERENCES

[1] Curtis AH. A cause of adhesions in the right upper quadrant. J Am Med Assoc 1930;94(16):1221–2.
[2] Fitz-Hugh T. Acute gonococcic peritonitis of the right upper quadrant in women. JAMA 1934;102(25):2094–6.
[3] Ricci P, Lema R, Sola V, et al. Fitz-Hugh-Curtis syndrome: three cases of incidental diagnosis during laparoscopy. J Obstet Gynaecol (Lahore) 2008;28(3):352–4.
[4] Faré PB, Allio I, Monotti R, Foieni F. Fitz-Hugh-Curtis syndrome: a diagnosis to consider in a woman with right upper quadrant abdominal pain without gallstones. Eur J Case Reports Intern Med 2018;5(2):000743.
[5] Sonavane A, Rathi P. Fitz-Hugh-Curtis syndrome. Indian J Med Res 2017;145(1):147.
[6] Reichert JA, Valle RF. Fitz-Hugh-Curtis Syndrome: A Laparoscopic Approach. JAMA J Am Med Assoc 1976;236(3):266–8.
[7] Woo SY, Il Kim J, Cheung DY, et al. Clinical outcome of Fitz-Hugh-Curtis syndrome mimicking acute biliary disease. World J Gastroenterol 2008;14(45):6975–80.
[8] Sam JW, Jacobs JE, Birnbaum BA. Spectrum of CT findings in acute pyogenic pelvic inflammatory disease. Radiographics 2002;22(6):1327–34.
[9] Wang CL, Guo XJ, Yuan ZD, et al. Radiologic diagnosis of Fitz-Hugh-Curtis syndrome. Chin Med J (Engl) 2009;122(6):741–4.