

## The broadened spectrum of Fitz-Hugh-Curtis syndrome

Fitz-Hugh-Curtis syndrome (FHCS) is a condition characterized by inflammation / infection of the liver capsule (perihepatitis) and adjacent peritoneal surfaces. It has, typically, been described in young women of child bearing age in association with either overt or latent pelvic inflammatory disease. Previously thought to be primarily attributable to *Neisseria gonorrhoeae*, of late, cases of FHCS due to *Chlamydia trachomatis* infection have been shown to outnumber those due to *Neisseria* by a ratio of 5:1. Although these organisms are regarded as the two major pathogens for the syndrome, other pathogens such as tuberculous and non-tuberculous mycobacteria, have been reported. Bacterial transmission to the liver may occur through hematogenous or lymphatic spread and in women from an infected fallopian tube via the right paracolic gutter. Clinically, the diagnosis of FHCS is made upon presentation of intense pleuritic type pain in the abdominal right upper quadrant (which may be referred to the right shoulder), accompanied by typical computed tomography (CT) findings of the abdomen. These findings consist of linear hepatic capsular enhancement in the arterial phase and perihepatic fat stranding in the portal phase (Fig. 1). The enhancement of the liver capsule is attributed to an increased blood flow or inflammation of the capsule. Although considered as a characteristic feature of the acute phase of FHCS. It may be seen in other conditions such as acute cholangitis, peritoneal carcinomatosis and systemic lupus erythematosus. Violin string-like adhesions between the liver and the parietal peritoneum have also been reported, particularly in the chronic phase of the disease.

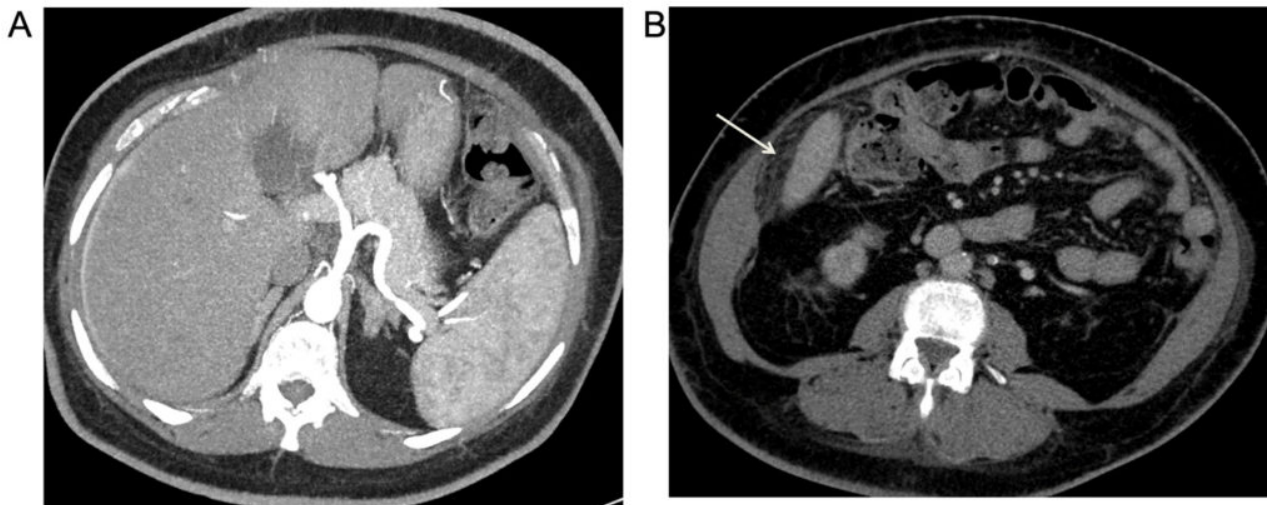


Fig. 1. A. Arterial phase CT scan shows linear enhancement at lateral surface of right hepatic lobe. B. Portal phase CT scan shows perihepatic fat stranding (arrow).

We have recently described a 58 year old woman with end stage renal disease due to diabetic nephropathy who was maintained on continuous ambulatory peritoneal dialysis (CAPD).

Her course was initially complicated by an exit site/tunnel infection due to methicillin sensitive *Staphylococcus aureus*. which responded to prolonged antibiotic treatment.

A month later, she developed recurrent peritonitis due to Coagulase negative *Staphylococcus*. The peritoneal dialysis catheter was removed and the patient transferred to hemodialysis. Post-operatively, a wound infection with abscess formation developed. The abscess was drained and cultures grew AmpC resistant *Enterobacter aerogenes*. Treatment with intravenous ciprofloxacin resulted in eradication of the wound infection.

Four weeks later, she complained of severe pleuritic type pain in the right upper abdominal quadrant. Computed tomography (CT) of the abdomen revealed linear hepatic capsular enhancement and perihepatic fat stranding (Fig. 1), thus establishing the diagnosis of FHCS. Gynecological examination excluded pelvic inflammatory disease. A three week course of ciprofloxacin resulted in complete resolution of her symptoms and CT findings.

Although, FHCS has traditionally been thought of as a disease affecting pre-menopausal women, it can occur in males and certain autoimmune and malignant conditions. This report further extends its spectrum describing its development in the context of peritoneal dialysis. Furthermore, it is the first time that AmpC *Enterobacter aerogenes* has been implicated as the causative organism in its pathogenesis. FHCS should be considered in the differential diagnosis of all patients presenting with pleuritic type right abdominal upper quadrant pain. Abdominal CT findings confirm the diagnosis.

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## **Publication**

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