F-18 FDG PET-CT in Fitz-Hugh-Curtis Syndrome

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Abstract

Positron emission tomography-computed tomography (PET-CT) has demonstrated its usefulness in evaluating nonspecific abdominal and inflammatory symptoms. We report a case of a young woman with chronic right upper quadrant abdominal pain. Fluorine-18 fluorodeoxyglucose PET-CT showed subhepatic hypermetabolism. Subsequent diagnostic laparoscopy confirmed the uncommon diagnosis of Fitz-Hugh-Curtis syndrome.

Keywords

► FDG
► PET-CT
► Fitz-Hugh-Curtis syndrome
► FHCS
► perihepatic uptake

Introduction

Positron emission tomography-computed tomography (PET-CT) has demonstrated its usefulness in the evaluation of nonspecific abdominal and inflammatory symptoms.1,2 The present case report discusses the fluorodeoxyglucose (FDG) PET-CT findings in a case of Fitz-Hugh-Curtis syndrome (FHCS). To our best knowledge, this is the first case describing such a syndrome diagnosed on FDG PET-CT.

Case History

A 29-year-old woman, with no known medical illnesses, presented with right upper quadrant (RUQ) pain and afebrile cough for 2 weeks. Esophagogastroduodenoscopy was unremarkable. Initial chest X-ray and CT showed minimal opacities with pleural changes at right lower lung base (► Fig. 1A) with no significant abnormalities in the abdomen (► Fig. 1B). Sputum culture subsequently grew *Mycobacterium szulgai*. In view of the initial radiographic findings, she was treated for a bacterial/ atypical community acquired pneumonia.

However, she continued experiencing RUQ pain with incipient anemia of inflammation. She was then referred for F-18 FDG PET-CT for further evaluation. PET-CT was obtained at 45 minutes after 194MBq of F-18 FDG was administered. The findings revealed curvilinear intense hypermetabolism at the subhepatic region (► Fig. 1C–D, arrows). Correlating with the initial CT, the focus corresponded to subtle subhepatic density (► Fig. 1E, arrow). No FDG avid abnormality was seen in the pelvis or both adnexae to suggest the presence of pelvic inflammatory disease (PID).

After the PET-CT, she was planned for diagnostic laparoscopy. Unfortunately, she was lost to follow-up due to travel restrictions of coronavirus disease 19 pandemic. She returned 5 months later with persistent pain, which had migrated to the left hypochondrium. She reported weight loss of approximately 10% in that time interval, but did not have fever, night sweats, or further respiratory symptoms. Repeat contrasted CT of the abdomen showed mild worsening of the subhepatic density (► Fig. 1F). Diagnostic laparoscopy revealed violin string-like adhesions at supracolic compartment involving the liver, gallbladder, duodenum,
and surrounding omentum (Fig. 1G). These features confirmed the diagnosis of FHCS.

A high vaginal swab culture and multiplex polymerase chain reaction assay grew *Candida spp* and detected *Ureaplasma parvum*, respectively. She was treated for both organisms but did not make a full clinical recovery. Bacterial and mycobacterial culture of omental tissue did not isolate any microorganism. She remains on follow-up to monitor her symptoms. Further laparoscopy for lysis of adhesions and repeat microbiologic diagnosis may be considered if her symptoms become progressive.

**Discussion**

FHCS is an uncommon inflammatory condition of the perihepatic capsule typically attributed to concomitant PID. As shown in this case, FHCS poses a diagnostic challenge because the patient usually presents with nonspecific RUQ pain, fever, and sometimes signs of salpingitis. The symptoms can mimic many other diseases (commonly, cholecystitis). Therefore, FHCS is usually diagnosed laparoscopically, where “violin string-like adhesions” are considered the hallmark of FHCS, especially in the chronic phase of the disease. There are no conclusive imaging features to diagnose FHCS. Perihepatic enhancement on CT is a common finding but is not diagnostic. This feature was not initially present in this patient. As F-18 FDG PET-CT can be used as an inflammatory imaging marker, the distribution of hypermetabolic focus at the subhepatic regions led to the suspicion of FHCS, which was subsequently confirmed laparoscopically.

FHCS is frequently caused by sexually transmitted pathogens such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. However, other causative pathogens have been reported. Despite appropriate antibiotic therapy, some patients may develop refractory pain, which is the case in this patient. In this patient, we postulate that the negative culture of laparoscopically biopsied tissue sample may be partly due to prior antibiotic administration and symptoms are due to ongoing aseptic inflammation. Although reports of nontuberculous mycobacteria causing FHCS are rare, we consider this a possible etiology in this patient because of the initial positive sputum culture and the absence of FDG avid abnormalities in the adnexae to suggest PID. Repeat laparoscopic assessment and lysis of adhesions may be considered in patients with chronic symptoms.

In conclusion, FHCS should be considered as a possible diagnosis when perihepatic uptake is demonstrated on F-18 FDG PET-CT in a female patient with RUQ pain.

**Conflict of Interest**

None declared.

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**Authors’ Contribution**

All authors contribute equally in conceptualizing and designing of the study, manuscript preparation, and final approval of the manuscript.

**Declaration of Patient Consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understand that her names and initials will not be published and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.
References