

Liver abscess as a rare complication of Crohn's disease: A case report

Crohn hastalığının nadir bir komplikasyonu olarak karaciğer absesi: Olgu sunumu

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Pyogenic liver abscess is a rarely seen extraintestinal complication of Crohn's disease. It has different features from other liver abscesses. Its clinical and laboratory findings are not specific and mimic the reactivation of Crohn's disease and diagnosis can be delayed. The radiological methods are very useful in diagnosis and treatment of liver abscess. In this paper, we present a patient with pyogenic liver abscess which developed in the course of Crohn's disease.

Key words: Liver abscess, Crohn's disease

Piyojenik karaciğer absesi Crohn hastalığının nadir görülen bir ekstraintestinal komplikasyonu olup, diğer karaciğer abselerinden farklı özellikleri mevcuttur. Klinik ve laboratuvar bulguları nonspesifiktir. Crohn hastalığının reaktivasyonu ile karışabilir ve tanı konulması da bu nedenle gecikebilir. Karaciğer absesinin tanı ve tedavisinde radyolojik metodlar oldukça yararlıdır. Bu yazıda, Crohn hastalığı seyrinde piyojenik karaciğer hastalığı gelişen bir hasta sunulmuştur.

Anahtar kelimeler: Karaciğer absesi, Crohn hastalığı

INTRODUCTION

Crohn's disease (CD) is a chronic inflammatory disease that can involve all portions of the gastrointestinal tract from mouth to anus. The gross mucosal lesions typically begin as aphthous ulcers. As the disease process advances, these ulcers enlarge, deepen, and eventually coalesce to form transverse and longitudinal linear ulcers. Lesions penetrate transmurally and involvement is usually segmental. The disease also has many extraintestinal manifestations. In the course of CD, hepatobiliary pathologies like pericholangitis, sclerosing cholangitis (4%), granulomatous hepatitis and cholelithiasis (25%) can be observed frequently (1). In addition, an infectious complication like pyogenic liver abscess (LA) can be seen rarely. Pyogenic LA usually develops secondary to biliary infections like cholecystitis, cholangitis or infection of organs that drain by portal vein (appendicitis, diverticulitis, inflammatory bowel disease). Despite the fact that portal pyemia is frequently found in inflammatory bowel disease, development of LA is rare. Incidence of pyogenic LA in CD is 114-297 per

100,000 and it is more often observed in younger ages (2). Sometimes LA is misdiagnosed as reactivation of CD because of shared symptoms and findings, and correct diagnosis can be delayed. Abdominal ultrasonography (USG) and computerized tomography (CT) are used most frequently in diagnosis of LA. The diagnosis can be confirmed by USG or CT guided percutaneous aspiration and drainage and then appropriate therapy can be planned according to culture and antibiogram (3).

In this paper we report a case of pyogenic LA that occurred in the course of Crohn's disease that was diagnosed and treated successfully by interventional radiological methods and medically.

CASEREPORT

A 54-year-old man was admitted to our unit with high fever, sweating and right upper abdominal pain. The complaints of abdominal pain and bloody diarrhea had appeared four months previously and he had been diagnosed as CD with ile-

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ocolonic localization, moderately active according to Harvey-Bradshaw clinical activity index. He was started on methylprednisolone (32 mg/day) and mesalamine tb (3x750 mg) (po). During the following weeks the steroid was tapered. A week previously, high fever, sweating and right upper abdominal pain appeared suddenly. He underwent abdominal CT with the suspicion of any complication that could be seen in the course of CD. CT revealed a cystic lesion 4x6x7 cm in the posterior segment of the right liver lobe, and caecal, ascendens colonic and terminal ileal diffuse wall thickness resulting in luminal narrowing (Figures 1,2).

The patient was in good health previously and was not taking any medications. He had been a heavy smoker for 35 years. He also had consumed alcohol (approximately 40 g/day) for 20 years. The patient was identified to be obese (body mass index 32) during physical examination. He had fever (37.7°C axillary temperature) and telangiectasia on his face. There were normal bowel sounds; however, right upper abdominal pain was noticed on palpation. No other pathological findings were detected in other systems. Laboratory results were as follows: erythrocyte sedimentation rate: 64 mm/hour, hemoglobin (Hb): 9.6 g/dl, mean corpuscular volume: 86 fL, leukocyte count: 8900/mm³, neutrophil count: 5290/mm³, platelet count: 410000/mm³, aspartate aminotransferase (AST): 19 U/L, alanine aminotransferase (ALT): 19 U/L, alkaline phosphatase (ALP): 170 U/L, gamma-glutamyl transpeptidase (GGT): 245 U/L, total bilirubin: 0.34 mg/dl, direct bilirubin: 0.10 mg/dl, total protein: 6.0 g/dl, albumin: 2.6 g/dl, gamma globulin: 0.8 g/dl, serum iron: 16 mcg/dl, total iron binding capacity: 222 mcg/dl, C-reactive protein: 16.3 mg/dl (normal <0.8), ferritin: 528 ng/ml (normal range: 25-320), and fibrinogen level: 1153 mg/dl (normal range: 200-400). Microscopic stool analysis showed moderately increased erythrocyte and leukocyte count. Abdominal ultrasonography revealed heterogen-hypoechoic cystic lesion in posterolateral segment of liver right lobe. The lesion had a sharp and irregular border in which there were nodular structures and septae. Serology of known hepatotropic viruses was negative. With these findings, diagnosis of liver abscess in the course of CD was established. Steroid therapy was terminated. Hemocultures were taken when the patient had high fever. Ceftriaxone (1x2 g/day) and metronidazole (2x500 mg) therapy was initiated before

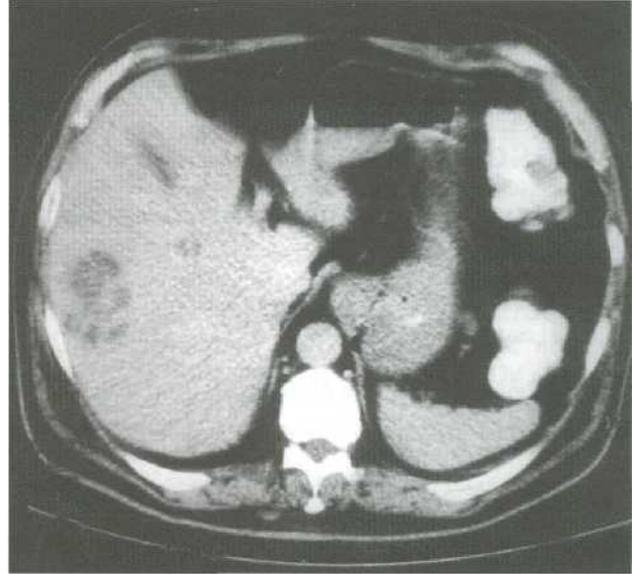


Figure 1. A cystic lesion 4x6x7 cm in size in posterior segment of right liver lobe.



Figure 2. Caecal, ascendan colonic and terminal ileal diffuse wall thickness resulting in luminal narrowing

planned abscess drainage. At the 4th day of anti-therapy, an USG-guided biopsy led to drainage of 70 ml of purulent fluid; a Gram's stain and cultures of the fluid showed no organism. Acid-fast organism was not disclosed by appropriate stain and culture. There was no growth of any organisms in the hemocultures. A total of 10 ml pus was drained during the following three days. Drainage tubes were withdrawn after USG control. The patient's condition improved. Persistence of high platelet count and high CRP levels suggested that the patient had an ongoing active CD. Colonoscopic examination revealed presence of discrete ulcers at ascending and transverse colon that are observed in patients with Crohn's colitis. Histological

examination of endoscopic biopsies confirmed CD. Mycobacterium tuberculosis was investigated in tissue by polymerase chain reaction (PCR) and was found negative. High GGT level was accepted as a result of alcohol intake. Low Hb was due to chronic disease. Antibiotherapy was changed from parenteral to oral on the 10th day and was stopped on 21st day of therapy.

DISCUSSION

Pyogenic LA is a rare complication of CD. It may be fatal, if left with no treatment or not treated early. LA is usually encountered in younger CD patients. LA incidence in CD is in between 114-297 per 100,000 and mean age of affected patients is approximately 36.5 years (2). It is known that some factors predispose to pyogenic LA. Intestinal mucosal ulcerations, destruction of normal mucosal integrity and invasion of portal venous system by microorganisms facilitate the reach of these pathogens to the liver. In the course of CD, increase in frequency of portal bacteriemia, perianal and enteric fistulae, perforations and subsequent intraabdominal abscesses, long-term steroid treatment, malnutrition, immunological abnormalities and surgical interventions are other predisposing factors to LA occurrence in CD (4, 5). Steroid treatment and destruction of normal mucosal integrity as a result of active intestinal mucosal lesions may have been the predisposing factors in our case. Patients usually present with signs and symptoms of high fever, chills, sweating, right upper abdominal pain, nausea-vomiting and weight loss that suggest CD reactivation. If diarrhea accompanies the symptoms above, the disease may be misdiagnosed as CD reactivation and hence steroid dose would be increased. Moreover, in some cases, surgical interventions that were performed in the situation of unresponsiveness to the treatment of high-dose corticosteroids fail (4, 6). LA development must be remembered in patients that have persistent pyrexia, and in this condition appropriate imaging studies must be performed as soon as possible (7). With the inception of the antibiotic era, the severe septic picture that was characterized by high fever, right upper abdominal pain and shock evolved to the milder picture of fever, blunt abdominal pain and adinamia. On physical examination, right upper quadrant pain is encountered in 50% of patients; hepatomegaly and icterus may also accompany. Icterus is usually together with severe illness and suppurative cholangitis. Sple-

nomegaly may be seen in chronic patients. Pleural effusion, empyema, atelectasia and pneumonia may rarely accompany. In our case, findings showed similarity to CD, but right upper abdominal pain alerted us. Rarely, CD may emerge as LA. Early diagnosis is very important in prevention of complications and in decreasing mortality. ESR increase, ALP elevation and leukocytosis can be seen. Mild elevation of GGT and ALT must also call our attention to LA. Growth of organisms occurs in 50% of hemocultures (8). In our patient, there was no growth in hemoculture; ALP, ALT and leukocyte count were normal despite high ESR. Normal ALP and ALT levels indicated that LA was not connected with bile ducts. Absence of leukocytosis can be explained with immunosuppression by steroid treatment. Positivity rate of direct culture of abscess approaches 90% (9). CT and USG localize abscess in liver; however, CT is superior to USG in determining the location and number of abscesses (10, 11). LA can be drained and cultured with USG or CT guidance. Percutaneous or endoscopic cholangiography is needed to localize cholangitic abscess. Abscesses are usually localized in right lobe of liver and may be multiple. In our patient, LA was confirmed with USG and CT, and it was unifocal and in the right lobe. The first step after confirmation of diagnosis is CT or USG guided drainage. Empiric antibiotherapy is begun till culture results are obtained. Microorganisms most often isolated in pyogenic LA are streptococcus and other aerobic/anaerobic streptococci, anaerobic Gram-negative bacillus, and aerobic Gram-negative bacillus, in order of frequency. It was reported that prolonged metronidazole treatment in CD with perianal complication could change the intestinal flora and cause streptococcal overgrowth (12). Absence of microorganism growth from pus may be related to previous antibiotherapy. Rate of mortality in pyogenic LA decreased to 16% with convenient antibiotherapy and effective drainage (13). Antibiotherapy should continue for 10-14 days. Persisting fever, polymicrobial growth in hemoculture, hyperbilirubinemia, pleural effusion, and elder age are factors demonstrating poor prognosis (14).

As a result, liver abscess is a rare complication of CD. Particularly in patients receiving steroid and antibiotics, we must remember and explore pyogenic LA in the occurrence of fever, chills, sweating, right upper quadrant pain, nausea, vomiting and weight loss.

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