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Liver Abscess Secondary to Sigmoid Diverticulitis ： A Case Report

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Abstract

Pyogenic liver abscess(PLA) which often presented by right sided abdominal pain and fever is a serious and life-threatening pathology. Biliary tract disease is the etiology of the abscess in most cases but sometimes the origin remains unidentified. A sigmoid septic source which maybe paucisymptomatic or hidden by an immunosuppressive treatment must be looked for. We present a case of liver abscess, which are secondary to unrecognized sigmoiditis. The etiologic diagnosis was made by abdomino-pelvic computed tomography, colonoscopy and barium-enema. The liver abscesses were emptied by aspiration and catheter drainage in conjunction with antibiotics. Surgical treatment of sigmoiditis was performed later. It is suggested that any liver abscess of unknown origin must lead to a search for unknown or disguised septic sigmoid pathology. Most of the time, contrast-enhanced abdomino-pelvic computed tomography makes the diagnosis possible, but barium enema or colonoscopy is sometimes necessary. (J Intern Med Taiwan 2005; 16: 289-294)

Key Words ： Liver abscess, Pyogenic, Sigmoiditis, Diverticulitis

Introduction

Liver is an organ that frequently develops abscess when infected, and the abscess maybe caused by bacterial, parasitic, or fungal infection. Liver abscess may develop from hematogenous spread of bacteria or local spread from contiguous sites of infection within peritoneal cavity. Previously, rupture of appendicitis with subsequent spread via portal system were the most common pathway to progress to liver abscess. However, the situation is relatively rare now because of advanced surgical management and

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application of antibiotic therapy[1]. Currently, liver abscess is most often associated with biliary tract disorders or pyogenic metastasis in a diabetic patient. Diverticulitis as the source of hepatic abscess is rare[2], especially sigmoid diverticulitis. Here, we present a unique patient with hepatic abscess secondary to sigmoid diverticulitis. The pathological and clinical features of the entity were briefly discussed.

Case Report

A 57-year-old male complained of fever, chills and general malaise for one week. He was sent to local medical clinic where liver abscess was suspected through abdominal computer tomography. Due to impending septic shock, he was transferred to Kaoshiung Military General Hospital for further evaluation and treatment. The patient has type 2 diabetes mellitus for more than ten years according his medical history.

Upon admission, he had fever up to 38.2 ℃ without tachycardia or tachypnea (BP 108/61 mmHg, PR 91/min, RR 18/min). The cardiopulmonary examination was normal and without organ enlargement but abdominal tenderness over right upper quadrant was noted. Jaundice and Murphy's sign were not found. The laboratory results were as follow: Hemogram: Hb 11.6 mg/dl, hematocrit 34.7%, leukocyte count 10600/UL, neutrophils 81.3%, lymphocyte 10.8%, eosinophils 17%; prothrombin time 13.4 seconds, CRP 17.2 mg/dl, total bilirubin 0.6 mg/dl. AST 27 IU/L, ALT 24 IU/L, ALP 115 IU/L, total protein 6.3 g/dl, Albumin 2.3 g/dl, Amebiasis Ab＜1 ：320. Other testing of parameters was within normal rage.

A chest x-ray was found to be normal. KUB was negative except lumbar spondylosis with spurring. Panendoscopy revealed esophageal, gastric & duodenal ulcers. The result of abdominal sonogram was compatible with liver abscess over S8 and gall bladder stone was also discovered. However, wall thickening and distention of gall bladder were not found so that acute cholecystitis was not considered.

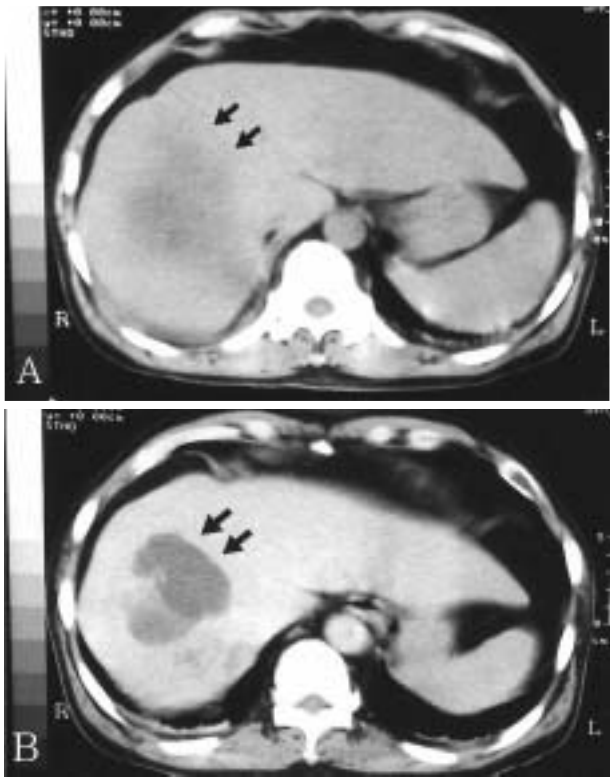

Fig.1A (Pre-contrast) : Lower attenuated perilesional edematous change.
B (Post-contrast) : Lobulated cystic-like lesion over S7 & S8 lobes can be consistent with liver abscesses.

Abdominal CT scan (Fig. 1) was arranged which revealed a large cyst, and an expansile separated lesion containing no air bubble with surrounding with edema over segment 7 & 8 of right liver. Percutaneous drainage was performed and empirical antibiotics of cefazolin and gentamycin were applied. The bacterial culture of abscess grew Prevotella melaninogenica (anaerobes), which was sensitive to cefoxitin. The regimen of antimicrobial drug was replaced to cefoxitin.

Lower abdominal pain and distension were noted two weeks later. Mild tenderness was noted over lower abdominal area. Abdominal sonography showed only residual abscess over segment 8. Sigmoidoscopy was performed up to sigmoid colon but only erythematous spots at rectum were found due to difficulty of further approach. However, colon lesion was still highly suspected.

Barium enema (Fig. 2) was performed and re-

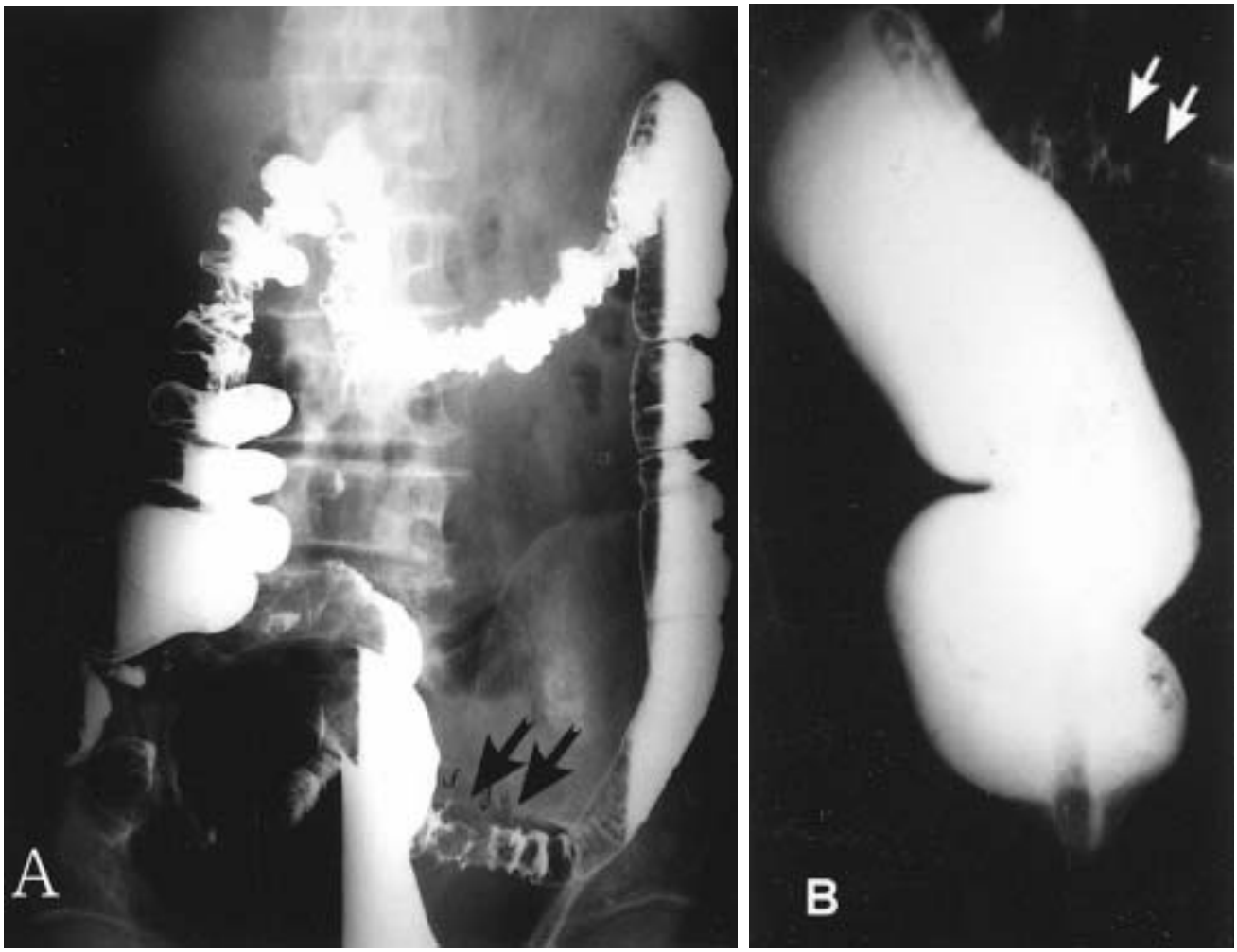

Fig.2A.Diverticula (black arrow) over sigmoid colon surrounding with segmental spasm and mucosal edematous changing.
B.Close view of diverticulitis (white arrow)

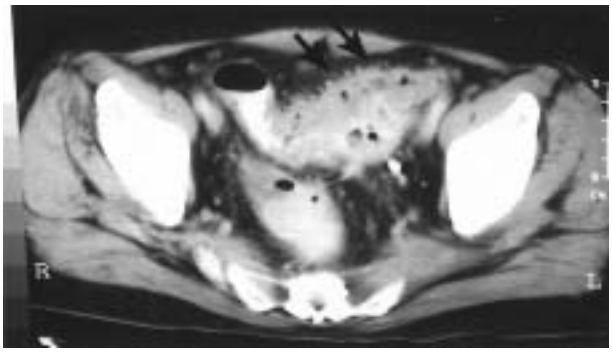

Fig.3.Thick mucosal fold over sigmoid colon surrounding with desmoplastic reaction of fat could be due to diverticulitis with rupture

vealed a segment of irregular mucosa and narrowing at the sigmoid colon. Focal colitis of the sigmoid colon was suspected. The sigmoid diverticulitis with liver abscess was highly suspected after consulting colon-rectal surgeon.

Another abdominal CT examination (Fig. 3) showed a noticeable irregular wall thickening over a long segment of transverse colon which might be caused by localized fluid accumulation and pelvic desmoplastic change. An Infectious colitis of sigmoid colon with regional abscess formation was exposed. Sigmoidoscopy was repeated and narrowed lumen still seen at sigmoid colon. The possibility of carcinoma infiltration or diverticulitis with micro-perforation needed to be clarified.

Explorative laparotomy revealed diverticulitis with adhesion to pelvic wall and anterior resection of sigmoid colon was carried out. Pathologic finding was diverticulosis (Fig. 4A) with some diverticula showing diverticulitis with pericolic abscesses (Fig. 4B). The result of bacterial culture of the micro-ab-

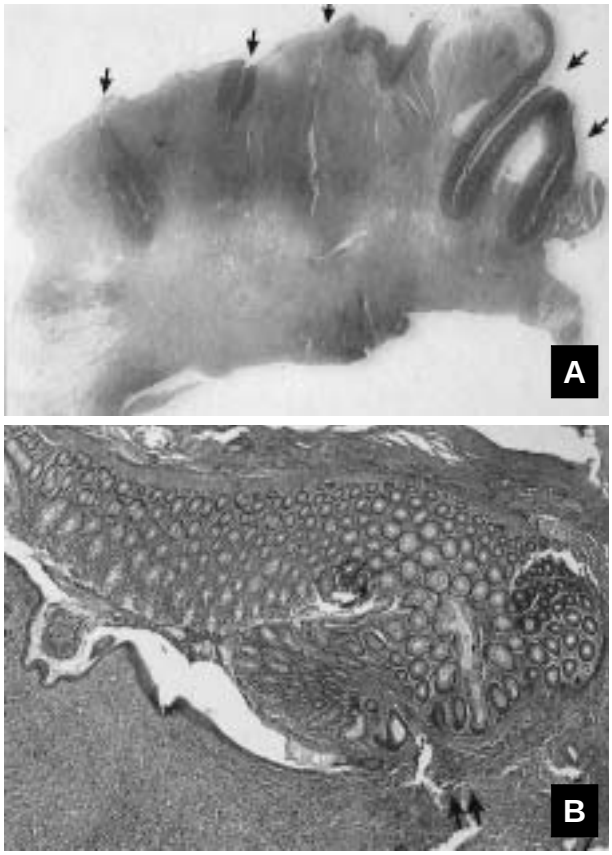

Fig.4A.Diverticulosis (black arrow) with multiple sac-like diverticula (x1).
B.Diverticulitis with mixed inflammatory cell infiltration. Perforation of diverticulum (black arrow) (x40)

scess was Prevotella melaninogenica (anaerobes) which was the same as the pathogen of liver abscess. Liver abscess secondary to sigmoid diverticulitis was diagnosed.

Discussion

Pyogenic liver abscess(PLA) remains a major diagnostic and therapeutic challenge despite advances in diagnostic technology and new strategies for treatment. Fever is the most common presenting sign of liver abscess. Nonspecific symptoms, such as chills, anorexia, weight loss, nausea, and vomiting, may develop. Only 50% of patients with liver abscesses, however, have hepatomegaly, right upper quadrant tenderness, or jaundice. Fever of unknown origin (FUO) may be the only presenting manifestation of liver abscess, especially in the elderly.

The liver receives blood from both the systemic and portal circulations. Increased susceptibility to infections would be expected given the increased exposure to bacteria. However, Kupffer cells lining the hepatic sinusoids clear bacteria so efficiently that infection rarely occurs. A pyogenic liver abscess can develop in various ways:

Biliary tract disease(e.g.: cholecystitis, cholangitis, stricture) remains the most common source of PLA. Obstruction of bile flow allows for bacterial proliferation. Through pressurization and distention of canaliculi, portal tributaries and lymphatics are invaded, with subsequent pylephlebitic abscess formation. With a biliary source, abscesses usually are multiple, unless they are associated with surgical interventions or indwelling biliary stents.

The portal venous system can be a source of abscess resulting from infections involving any organ drained by this system. Appendicitis was the leading source of PLA in the preantibiotic era, but it essentially has been eliminated in recent years. The association through which this is thought to occur is pylephlebitis (septic thrombophlebitis) of any tributary of the portal venous system. Infections in organs in the portal bed can result in a localized septic thrombophlebitis. Septic emboli are released into the portal circulation, trapped by the hepatic sinusoids, and become the nidus for microabscess formation. Thus, abscesses initially are multiple but usually coalesce into a solitary lesion.

Microabscess formation due to hematogenous dissemination of organisms can be seen in association with illness that involves systemic bacteremia, such as endocarditis and pyelonephritis.

Contiguous spread from localized infection of the gallbladder and the perihepatic space can result in PLA. Abscesses can result from fistula formation between local intraabdominal infections.

Penetrating hepatic trauma can inoculate organisms directly into liver parenchyma, resulting in PLA. Nonpenetrating trauma results in localized hep-

atic necrosis, intrahepatic hemorrhage, and bile leakage due to disruption of canaliculi. The resulting tissue environment permits bacterial growth with resultant PLA. Lesions of this etiology typically are solitary in nature.

PLA has been reported as a secondary infection of amebic abscess, hydatid cystic cavities, and metastatic and primary hepatic tumors. It also has been a complication of liver transplantation, hepatic artery embolization in the treatment of hepatocellular carcinoma, and the ingestion of foreign bodies, which penetrate the liver parenchyma.

Most of the patients with Klebsiella pneumoniae liver abscess were diabetic and without biliary tract disease[7]. Diabetes is known to interfere with neutrophil chemotaxis and phagocytetosis, but its influence on the function of macrophages, including Kuffer's cells, is still unknown. However, if the function of Kufffer's cells is also impaired in diabetic patients, the preponderance of K. pneumoniae liver abscess cases in the population could be explained by the escape of enteric K. pneumoniae from phagocytosis by Kuffer's cells[10].

In this case, haematogenous or contiguous paths were highly suspected. The portal circulation distributes to various areas of the bowel and several illness are caused through portal circulation system (e.g. appendicitis can lead to thrombophlebitis of vessels of the portal circulation). Septic emboli may accumulate in liver, resulting in abscess formation.

Pyogenic liver abscess is usually a polymicrobial infection that has ascended from the gastrointestinal tract. In Western countries, the most frequent aetiological agents of pyogenic liver abscess were Escherichia coli, streptococci, and anaerobic bacteria. However, over the past two decades in Taiwan, liver abscess has usually been caused by a single microorganism, Klebsiella pneumoniae, presenting in 50 ―88% of pyogenic liver abscesses[3,6,10]. Previous studies from Taiwan demonstrated that diabetes mellitus is the most common underlying condition, with a prevalence ranging from 45% to 75% in patients with K pneumoniae liver abscess[3,7,8,10].

The pathogenic role of anaerobes was under-appreciated until the isolation of anaerobes from 45% of cases of PLA was reported in 1974. The most frequently encountered anaerobes are Bacteroides species, Fusobacterium species, and microaerophilic and anaerobic streptococci[9]. A colonic source is usually the initial source of infection.

Staphylococcus aureus abscesses usually result from hematogenous spread of organisms involved with distant infections, such as endocarditis. Entamoeba histolytica, that causes amebiasis, is an intestinal infection. The organism is carried through the blood to the liver where the abscess is formed. Patients may or may not have symptoms of intestinal infection concurrently with liver abscess. Fungal abscesses primarily are due to Candida albicans and occur in individuals with prolonged exposure to antimicrobials, hematologic malignancies, solid-organ transplants, and congenital and acquired immunodeficiency.

The major pathogens of infectious emboli in the portal system causing pyogenic liver abscess are polymicrobial or anaerobes reflecting the gastrointestinal flora involved in the primary infection. Frequent causes of anaerobic liver abscess are acute and chronic inflammatory bowel disease with or without perforation, malignancy and/or surgery of the gastrointestinal tract or pelvic organs, and biliary tract disease[4]. In this case, Prevotella melaninogenica (anaerobes) was cultured from both of liver abscesses and sigmoid diverticulitis. Liver abscesses secondary to sigmoid diverticulitis was further confirmed by the culture result.

In conclusion, it is important to be aware of sigmoid diverticulitis as a possible cause of liver abscess. Furthermore, since asymptomatic sigmoid diverticulitis can cause liver abscesses, all patients who present with the triad of fever, jaundice and an enlarged tender liver should probably have a barium enema or

colonoscopy to rule out this possibility. Once the diagnosis is considered, proper treatment should include prompt drainage of the abscesses, treatment of the locally inflamed sigmoid colon and administration of high doses of antibiotics based on the cultures of pus obtained at surgery[5].

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乙狀結腸憩室炎併發肝膿瘍：一病例報告

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摘 要

化膿性肝膿瘍經常以右上腹痛及發燒來表現，常見的原因大部分以膽道疾病爲主，但有時也原因不明，此時腸道疾病所造成的併發症就必須列入鑑別診斷。一個化膿性肝膿瘍的案例，經抗生素治療後兩週，發現有腹脹及下腹痛的情形，最後經由腹部電腦斷層及鋇劑灌腸攝影，診斷出乙狀結腸憩室炎，經手術治療後發現培養出的細菌與之前化膿性肝膿瘍的菌種相同，最後診斷爲乙狀結腸炎併發肝膿瘍。經由此一病例，對於任何不明原因所造成的化膿性肝膿瘍，安排腸道的檢查：包括大腸鏡、鋇劑灌腸及腹部電腦斷層等檢查，有其必要性，以排除腸道疾病所造成的併發症的可能性。