CASE REPORT

Two Cases of Liver Abscesses Derived from Dental Disease in Patients with Alcoholic Chronic Pancreatitis

Yukiko Osawa, Hiroyuki Isayama, Suguru Mizuno, Yousuke Nakai, Miho Matsukawa, Dai Mohri, Hirofumi Kogure, Takashi Sasaki, Natsuyo Yamamoto, Kenji Hirano, Naoki Sasahira, Minoru Tada and Kazuhiko Koike

Abstract

Among the etiologies of pyogenic liver abscess (PLA), bacterial spread from the biliary tract or portal flow is the major cause, while the onset of PLA due to arterial bacterial transmission is rare. We herein report two cases of PLA thought to be caused by arterial transmission from dental disease. In both cases, there was benign biliary stricture as a result of alcoholic chronic pancreatitis, although normal oral flora was detected as the causative bacteria and oral hygiene was poor in both patients. We presumed that the origin of PLA was dental disease and successfully treated the patients with percutaneous drainage, antibiotics and dental procedures.

Key words: pyogenic liver abscess, dental disease, alcoholic chronic pancreatitis

(Intern Med 54: 1623-1625, 2015) (DOI: 10.2169/internalmedicine.54.4043)

Introduction

Liver abscesses are classified as bacterial or amebic. The major etiology of bacterial liver abscesses is bacterial spread from the biliary tract and/or bacterial transmission via the portal flow, whereas the development of a pyogenic liver abscess (PLA) due to hematogenous bacterial transmission is reported to be rare (1, 2). It has been reported that dental infection and/or procedures are a possible origin of pyemia; however, there are only a few reports of PLA with a dental origin occurring via hematogenous spread.

Patients with alcoholic chronic pancreatitis (ACP) are immunocompromised, as they often have diabetes mellitus and malnutrition. Moreover, alcohol consumption is reported to be a risk factor for dental infection; therefore, patients with ACP are at high risk for PLA with a dental origin.

In this paper, we present two cases of PLA in patients with ACP successfully treated with percutaneous abscess drainage and antibiotics. Although both patients had a history of biliary interventions for benign biliary stricture, cultures of the abscesses confirmed hematogenous spread from dental infections, not a biliary etiology.

Case Reports

Case 1

A 60-year-old man with ACP was admitted to our hospital due to a fever and disturbance of consciousness. He had consumed 90 g of alcohol per day and smoked 20 cigarettes per day for 44 years. He had previously diagnosed with chronic pancreatitis with pancreatic stones and undergone choledoho-duodenostomy for benign distal biliary stricture. He was currently on insulin therapy for pancreatic diabetes; however, the control was poor, with an HbA1c (NGSP) level of 10.8% on admission.

The patient was in a state of shock, and his vital signs were as follows: a consciousness level of E4V4M6 according to the Glasgow Coma Scale, a blood pressure of 80/52 mmHg, a heart rate of 110 beats/min and a body temperature of 37.8°C. On a physical examination, he was thin [body mass index (BMI), 19.8 kg/m²] and displayed right lateral abdominal tenderness. Laboratory tests showed an
elevated inflammatory reaction [white blood cell count (WBC), 20,200/μL; C-reactive protein (CRP) level, 23.17 mg/dL] and acute kidney injury (serum creatinine, 3.33 mg/dL). Elevated liver function parameters were also noted [aspartate aminotransferase (AST), 953 IU/L; alanine aminotransferase (ALT), 463 IU/L; gamma-glutamyl transpeptidase (GGTP), 98 IU/L; alkaline phosphatase (ALP), 1,254 IU/L]. Computed tomography (CT) revealed multiple low-density areas in segment 6 of the liver, with a maximum diameter of 55 mm, although the biliary system was not dilated (Fig. 1). We administered 2.25 g of piperacillin/tazobactam q.i.d., and the patient underwent percutaneous abscess drainage using an 8-Fr catheter. Streptococcus sanguis, known to be a member of the normal oral flora, was detected on an abscess fluid culture. A consultation with a dentist revealed severe periodontitis, and the patient received treatment for decayed teeth. Endoscopic retrograde cholangiography (ERC) via the choledocho-duodenostomy revealed no communication between the abscess and biliary tree. The patient fully recovered after four weeks of antibiotic therapy.

In both of the two reported cases, the causative bacteria of PLA were known to be species of the normal oral flora. In addition, both patients had decayed teeth and severe periodontitis. We therefore presumed that the bacterial origin of these liver abscesses was dental disease and that the bacteria had been transmitted to the liver via the arterial flow.

A total of 11 cases of PLA derived from dental disease reported between 1987 and 2013, based on our search of PubMed (Table) (3-9). Fusobacterium and Streptococcus are reported to be major species of causative bacteria of PLA arising from dental infection and were also detected on abscess fluid cultures in our cases; these bacteria are also known to be normal oral flora.

Among the etiologies of PLA, biliary tract disease is reported to be the most frequent (15.5-37%), with portal pyemia being the second (11-15%), and the rate of a hematogenous origin is reported to be 6-13.5% (1, 2). In fact, both of our patients had significant potential to develop cholangitis. Patient 1 had a history of choledocho-duodenostomy, and patient 2 had undergone plastic stent placement for distal benign biliary stricture due to ACP and currently used insulin for the treatment of pancreatic diabetes, although the control was poor, with an HbA1c (NGSP) level of 8.3%.

Discussion

Case 2

A 42-year-old man with ACP was admitted to our hospital due to a fever. He had consumed 60 g of alcohol per day and smoked 20 cigarettes per day for 23 years. He had previously undergone plastic stent placement for distal benign biliary stricture due to ACP and currently used insulin for the treatment of pancreatic diabetes, although the control was poor, with an HbA1c (NGSP) level of 8.3%.

The patient was also thin (BMI, 18.0 kg/m²) and had a fever (body temperature, 38.7°C). He exhibited tenderness around the umbilicus, and laboratory tests showed an elevated inflammatory reaction (WBC, 11,600/μL; CRP level, 13.84 mg/dL). Liver function parameters were within the normal limits (AST, 15 IU/L; ALT, 9 IU/L; GGTP, 85 IU/L; ALP, 528 IU/L), and the bilirubin level was also normal (total bilirubin, 0.3 mg/dL). Meanwhile, contrast-enhanced CT revealed a low-density lesion with peripheral enhancement in the caudate lobe of the liver, which measured 34 mm in diameter (Fig. 2). There was no evidence of dilatation of the biliary tract.

We administered 1.5 g of ampicillin/sulbactam iv t.i.d., and the patient underwent percutaneous abscess drainage using an 8-Fr catheter. Fusobacterium necrophorum, known to be a member of the normal oral flora, was detected on both blood and abscess fluid cultures. A consultation with a dentist revealed severe periodontitis, and the patient received treatment for decayed teeth. ERC revealed no communication between the abscess and biliary tree. The patient fully recovered after four weeks of antibiotic therapy.
placement across the site of biliary stricture. However, in both cases, imaging studies showed no dilatation of the biliary system, a typical finding for cholangitis. In addition, the causative bacteria were not enteric bacteria, which are the most frequent causative bacteria of cholangitis, although these species are known to be normal oral flora. In addition, both patients had poor oral hygiene. Therefore, we presumed that bacterial transmission from the oral cavity to the liver via the arterial flow was the cause of the liver abscesses, although the involvement of a biliary tract infection cannot be excluded.

An immunocompromised status, such as that associated with diabetes mellitus and malignancy, is reported to be a risk factor for PLA (10). However, among previously reported cases of PLA derived from dental disease, only two of 11 patients had an immunocompromised status (Table), including one case of dysfunction of B-cells and one case of diabetes. Dental infection itself may be a cause of PLA, even in immunocompetent patients. Patients with ACP often have diabetes mellitus simultaneously, and diabetes is reported to be a risk factor for gingivitis and periodontitis (11). Moreover, alcohol consumption is also known to be associated with periodontitis (12), and patients with ACP tend to be immunocompromised due to malnutrition. Therefore, patients with ACP are at significantly high risk for developing PLA derived from dental infection.

We experienced two rare cases of PLA derived from dental infection in ACP patients. Dental infection should be considered as a potential origin of PLA in patients with ACP.

The authors state that they have no Conflict of Interest (COI).

Table. Published Cases of Pyogenic Liver Abscesses Infected from Dental Disease.

| Reference | Age | Gender | Organisms                          | Underlying condition                  | Treatment                | Outcome |
|-----------|-----|--------|------------------------------------|--------------------------------------|--------------------------|---------|
| 3         | 29  | M      | F. nucleatum                       | Dysfunction of B cell                 | PTAD, antibiotics        | Cured   |
| 3         | 54  | F      | F. nucleatum, S. viridans, A. odontolyticus | N/P                                  | N/A                      | N/A     |
| 3         | 69  | M      | F. nucleatum                       | N/P                                  | N/A                      | N/A     |
| 3         | 59  | M      | F. nucleatum                       | N/P                                  | PTAD, antibiotics        | Cured   |
| 4         | 60  | M      | S. anginosus                       | COPD                                 | Antibiotics              | Cured   |
| 4         | 39  | N/A    | S. intermedius                     | N/P                                  | PTAD, antibiotics        | Cured   |
| 5         | 59  | F      | F. varium, P. melaninogenica, B.vulgaris | N/P                                  | None                     | Died    |
| 6         | 25  | M      | F. nucleatum, P. species           | Hepatitis B                          | PTAD, antibiotics        | Cured   |
| 7         | 18  | M      | S. intermedius                     | N/P                                  | PTAD, antibiotics        | Cured   |
| 8         | 40  | M      | F. nucleatum                       | DM, diverticulum                     | One puncture, antibiotics | Cured   |
| 9         | 36  | M      | F. nucleatum                       | N/P                                  | PTAD, antibiotics        | Cured   |
| Our case 1| 60  | M      | F. nucleatum                       | ACP, DM                               | PTAD, antibiotics        | Cured   |
| Our case 2| 42  | M      | S. sauguis                         | ACP, DM                               | PTAD, antibiotics        | Cured   |

N/A: not available, N/P: nothing particular, COPD: chronic obstructive pulmonary disease, ACP: alcoholic chronic pancreatitis, DM: diabetes mellitus, PTAD: percutaneous transhepatic abscess drainage

References

1. Seeto RK, Rockey DC. Pyogenic liver abscess. Changes in etiology, management, and outcome. Medicine (Baltimore) 75: 99-113, 1996.
2. Mangukiya DO, Darshan JR, Kanani VK, Gupta ST. A prospective series case study of pyogenic liver abscess: recent trends in etiology and management. Indian J Surg 74: 385-390, 2012.
3. Kajiya T, Uemura T, Kajiya M, et al. Pyogenic liver abscess related to dental disease in an immunocompetent host. Intern Med 47: 675-678, 2008.
4. Giuliano S, Rubini G, Conte A, et al. Streptococcus anginosus group disseminated infection: case report and literature review. Infez Med 3: 145-154, 2012.
5. Ohyama H, Nakasho K, Yamanegi K, et al. An unusual autopsy case of pyogenic liver abscess caused by periodontal bacteria. Jpn J Infect Dis 62: 381-383, 2009.
6. Lei WY, Chang WH, Shih SC, Liu CJ, Shih CH. Pyogenic liver abscess with Prevotella species and Fusobacterium necrophorum as causative pathogens in an immunocompetent patient. J Formos Med Assoc 108: 253-257, 2009.
7. Neumayr A, Kubiz R, Bode JG, Bilk P, Haussinger D. Multiple liver abscesses with isolation of Streptococcus intermedius related to a pyogenic dental infection in an immune-competent patient. Eur J Med Res 15: 319-322, 2010.
8. Nozawa Y, Yoshita S, Fukushima M, et al. A case of pyogenic liver abscess infected with Fusobacterium necrophorum depicted by microscopy and confirmed by tissue culture. Intern Med 50: 1815-1819, 2011.
9. Yoneda M, Kato S, Mawatari H, et al. Liver abscess caused by periodontal bacterial infection with Fusobacterium necrophorum. Hepatol Res 41: 194-196, 2011.
10. Kaplan GG, Gregson DB, Laupland KB. Population-based study of the epidemiology of and the risk factors for pyogenic liver abscess. Clin Gastroenterol Hepatol 2: 1032-1038, 2004.
11. Kim EK, Lee SG, Choi YH, et al. Association between diabetes-related factors and clinical periodontal parameters in type-2 diabetes mellitus. BMC Oral Health 12: 64, 2013.
12. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol 2000 62: 59-94, 2013.