Morganella morganii causing solitary liver abscess complicated by pyopericardium and left pleural effusion in a nondiabetic patient

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Morganella morganii is a rare cause of solitary liver abscess in Taiwan. The complication of pyopericardium and pleural effusion in nondiabetic patient with solitary liver abscess are also rare. We present a case of a 48-year-old nondiabetic woman who experienced with epigastric discomfort 1 month prior to admission. Chills and fever developed 2 weeks before admission. Physical examination on admission revealed engorgement of the jugular vein over the right neck, precordial friction rubs, and tenderness over the right upper quadrant of abdomen. Chest film showed mild cardiomegaly and left pleural effusion. Computed tomography of the abdomen showed liver abscess, left hepatic lobe, pyopericardium, and left pleural effusion. M. morganii was isolated from 2 sets of blood cultures, one set of hepatic pus culture, and one set of pericardial pus culture. After pigtail drainage of liver abscess, pyopericardium for 12 days, and ceftriaxone intravenous administration for 19 days, the patient was discharged in stable condition.

Key words: Liver abscess, Morganella morganii, pleural effusion, pyopericardium

Pyogenic liver abscess is usually polymicrobial because of the ascending route of infection from the gastrointestinal tract [1]. Over the past 15 years in Taiwan, liver abscess caused by a single pathogen, Klebsiella pneumoniae, occurred in diabetic patients without intraabdominal or biliary tract infection [2]. Pyogenic liver abscess is a potentially lethal condition. Early diagnosis is essential to reduce mortality or morbidity. Modern imaging techniques such as ultrasonography and computed tomography are sensitive and noninvasive diagnostic tools that can provide valuable information in diagnosing liver abscess [3]. A higher proportion of K. pneumoniae has been reported as an etiologic agent in pyogenic liver abscesses in Taiwan compared with western populations [4,5]. However, Morganella morganii is a rare pathogen of pyogenic liver abscess in Taiwan. We present a case of solitary liver abscess caused by M. morganii and complicated by pyopericardium and left pleural effusion, which are rare complications of liver abscess in nondiabetic patient. This case may also serve as a reminder that delayed diagnosis and institution of appropriate antimicrobial therapy for liver abscess may result in severe complications.

Case Report

A 48-year-old woman from Tainan County, the southern part of Taiwan, was admitted due to epigastric discomfort, which had begun 1 month prior to admission. She received a prescription for antacid at the outpatient department of a regional hospital but did not respond. Chills and fever developed 2 weeks before she visited another regional hospital, where liver abscess with extension to the pericardial cavity was found. Pericardiocentesis with drainage catheter retention and liver abscess drainage were performed. Due to a lack of available beds in the intensive care unit, she was transferred to Armed Forces Tsouing Hospital in Kaohsiung. At admission, she had clear consciousness. Physical examination showed an acutely ill patient with body temperature of 37.6°C; blood pressure 80/40 mm Hg; pulse rate 126 per min; and respiratory rate 34 per min. Engorgement of jugular vein over the right neck, precordial friction rubs, and tenderness over the right upper quadrant of abdomen were noted. Laboratory study showed a white blood cell count of 22 000 /mm³; neutrophil/lymphocyte 95/4; hemoglobin 11.7 g/dL; and platelet count 680 000 /mm³. Serum biochemistry showed aspartate transaminase 179 IU/L (normal range, 7-30 IU/L); alanine transaminase 95 IU/L (normal range, 7-30 IU/L); blood urea nitrogen 11 mg/dL; creatinine 1.1 mg/dL; glucose 100 mg/dL; Na 141 mEq/L; and K 4.6 mEq/L. Chest film showed mild
cardiomegaly and left pleural effusion. Computed tomography of the abdomen showed a liver abscess in the left hepatic lobe, pyopericardium, and left pleural effusion (Fig. 1). Pleurocentesis was performed and pleural effusion with a xanthochromic appearance was collected. The sample showed pleocytosis with red cells of 100 /μm³; white cells 13 330 /μm³; neutrophil/lymphocyte 40/60; protein 1.7 g/dL; and lactate dehydrogenase 198 IU/dL. No bacteria was found on gram stain of pleural effusion and culture of pleural effusion was negative. Two sets of blood cultures, one set of hepatic pus culture, and one set of pericardial pus culture grew M. morganii, which was susceptible to ceftriaxone, ceftazidime, imipenem, gentamicin, amikacin, piperacillin, piperacillin-tazobactam, and cefepime, but resistant to first- and second-generation cephalosporins. After pigtail drainage of the liver abscess, pyopericardium for 12 days, and ceftriaxone 2 g intravenously daily for 19 days, the clinical response was well. Laboratory studies showed white blood cell, aspartate transaminase, and alanine transaminase returned to normal range. The patient was discharged in stable condition with oral cefixime 200 mg 2 times a day for 30 days and followed up regularly at outpatient department. Follow-up abdominal sonography and echocardiogram showed no residual abscess or pyopericardium.

Discussion

Pyogenic liver abscess is usually a complication of biliary tract disease (30%–35%), contiguous infections such as subphrenic abscess or empyema of gallbladder (15%) and intestinal disease (15%) [6]. Escherichia coli is the most common liver abscess pathogen, with a percentage of about 35% to 45% worldwide [7]. However, these disease characteristics have been undergoing changes in Taiwan. K. pneumoniae has emerged as the leading liver abscess pathogen in Taiwan, with the percentage rising from 30% in the 1980s to over 80% in the 1990s [8]. Some patients experience serious extrahepatic complications such as endophthalmitis, meningitis, lung abscess, and necrotizing fasciitis [9,10]. In addition to antibiotic therapy, intensive percutaneous drainage or surgical drainage is needed. Percutaneous drainage under sonographic or computerized tomographic guidance has been suggested as the first choice of treatment for patients with pyogenic liver abscess [11-14].

M. morganii is a gram-negative, aerobic, facultative anaerobic rod. The genus Morganella has been included in the tribe Proteae of the family Enterobacteriaceae together with 2 other genera, Proteus and Providencia [15]. The Morganella were originally placed in the genus Proteus until DNA studies conducted by Brenner et al [15] concluded that they warrant their own separate genus since they were no more closely related to the protein than to any other member of this family. Currently, only a single species, M. morganii, is known to exist. This bacteria grows well on MacConkey agar with clear, lactose-negative colonies. It produces phenylalanine deaminase and grows in potassium cyanide [16]. It can establish itself as part of the resident flora within a hospital and can infect a large number of patients. The urinary tract, especially in old and catheterized patients, is most commonly involved, but sepsis, pneumonia, chorioamnionitis, wound infections [17], pericarditis [18], spontaneous bacterial peritonitis [19], and chylothoangreenosom [20] may also occur. Most isolates (97%) are urease positive [21] and are resistant to multiple antibiotics [22,23]. In one study, 34% of 220 routine fecal specimens contained M. morganii, suggesting that the source of infecting organisms was the gastrointestinal tract [21]. This species was also implicated as a cause of gastroenteritis [21]. The liver has a dual blood supply: sterile arterial blood from the hepatic artery and venous blood from the gut. Transient bacteremia of the portal system is not unusual [2]. Therefore, the most probable source of M. morganii in this case of liver abscess was from the gut. Species of the family Enterobacteriaceae, including Citrobacter freundii, Enterobacter cloacae, M. morganii, Serratia marcescens, and Yersinia enterocolitica are naturally resistant to aminopenicillins and early cephalosporins. This resistance phenotype is mediated by chromosomally encoded β-lactamases (AmpC) [24]. A plasmid-mediated AmpC β-lactamase resistance to

Fig. 1. Computed tomography of abdomen shows liver abscess, left hepatic lobe (arrow), pyopericardium (arrow) and left pleural effusion (arrow).
cephamycin and extended-spectrum cephalosporins has been found [25]. The production of extended spectrum β-lactamase (ESBL) in *M. morganii* has also been reported [26,27].

β-Lactamase production is the most common mechanism of bacterial resistance to β-lactam antibiotics [28]. Resistance to β-lactams is usually related to the production of AmpC β-lactamase and ESBL. First-generation cephalosporins, cefoxitin, and ampicillin are strong inducers of AmpC β-lactamase and are hydrolyzed rapidly. Consequently, they induce their own destruction and thus lack activity against inducible species [29]. Clavulanate does not effectively inhibit class I enzyme [29]. Thus, the use of the above antibiotics to treat *M. morganii* infection is not appropriate.

The combination of tazobactam with piperacillin shows highly synergistic effect and can be active against species that are commonly inducible for class I β-lactamase [30]. Many third-generation cephalosporins and ureidopenicillins remain active against β-lactamase-inducible strains but not against stably derepressed mutants that produce a large amount of enzyme without induction [31]. Stably derepressed mutants for chromosomal class I β-lactamase are produced at high frequency by inducible strains, and may be selected during therapy, sometimes resulting in clinical failure [31]. Fourth-generation cephalosporins and carbapenem may be more effective against stably derepressed mutants for chromosomal class I β-lactamase [31]. In this study, the *M. morganii* isolate was susceptible to ceftriaxone, ceftazidime, imipenem, gentamicin, amikacin, piperacillin, piperacillin-tazobactam, and cefepime, but resistant to first- and second-generation cephalosporins. In Taiwan, a first-generation cephalosporin is commonly used for patients with *Klebsiella* liver abscess, with a duration of up to 4 weeks. Moreover, gentamicin or amikacin is added to the regimen during the first week of treatment [5]. As for the endophthalmitis or meningitis complicating from *Klebsiella* liver abscess, a third-generation cephalosporin (ceftriaxone) in addition to percutaneous drainage is required [5].

*M. morganii* often causes opportunistic infection, but liver abscess complicated by pyopericardium and left pleural effusion due to this organism has not been previously reported. It is difficult to explain for the rarity of liver abscess due to *M. morganii* despite its presence as a part of the flora of the intestinal tract. This patient was a relatively immunocompetent woman. She had robust health in the past, except for the development of pulmonary tuberculosis 5 years ago. Pyopericardium may have developed due to liver abscess rupture into the pericardium and left pleural effusion may have been due to reactive changes of associated with pyopericardium [32]. In conclusion, pyogenic liver abscess due to *M. morganii* is very rare and clinical diagnosis is not possible. Broad-spectrum antibiotics active against aerobic organisms remain an excellent choice for treatment before definitive recognition of *M. morganii* and antimicrobial susceptibility antibiotic are available. The favorable outcome in this patient may be explained by the lack of a mixed infection, and by the early isolation of the organism leading to appropriate antibiotic therapy. Delayed diagnosis may lead to serious complications which are preventable by complete history-taking, physical examination, and alertness to the possibility of liver abscess.

References


