Pyogenic liver abscess (PLA) is not a common disease in children, with an incidence of 10/100,000 hospitalizations. In children with PLA, *Staphylococcus aureus* is the most common pathogenic organism. Recently, *Klebsiella pneumoniae* has been increasingly found as a PLA pathogen. PLA due to this bacterium often leads to formation of extrahepatic abscesses. The treatment of choice is dual therapy with insertion of percutaneous catheter drainage and antibiotic therapy. We report 2 cases of PLA due to *K. pneumoniae* in immunocompetent children. We successfully treated patient 1 with percutaneous catheter drainage for 18 days and 6-week course of antibiotic therapy. Patient 2 was treated with percutaneous needle aspiration and antibiotic therapy for the same period. In both patients, the PLAs showed the ultrasound-confirmed resolutions after the dual therapy.

**Key words:** Anti-Bacterial Agents; Drainage; Immunocompetence; *Klebsiella pneumoniae*; Liver abscess, Pyogenic

**Introduction**

Pyogenic liver abscess (PLA) is not a common disease in children, with an incidence of 10/100,000 hospitalizations. In children with PLA, *Staphylococcus aureus* is the most common pathogenic organism. Recently, there has been an increasing incidence of PLA due to *Klebsiella pneumoniae* in Korea.

This disease entity has been found to be caused by one of the following: bacteria entering portal circulation; primary bacteremia; and cryptogenic infection. Although the improved diagnostic modalities and antibiotic therapy have decreased mortality, PLA remains a fatal disease with mortality rates ranging from 8.2% to 16.8%. Immunocompromising diseases, such as chronic granulomatous diseases, diabetes mellitus or malignancy, and preexisting hepatobiliary or pancreatic diseases predispose children to PLA. Despite such risk factors, we recently experienced 2 immunocompetent children with PLA. In this case report, we report the 2 cases of successfully treated PLA due to *K. pneumoniae* in the children.
Case

1. Case 1

A previously healthy 12-year-old boy visited our hospital emergency department (ED) with fever and right upper quadrant (RUQ) pain lasting for 6 days without antibiotic therapy. He also complained of nausea and vomiting. The initial temperature was 39.6°C. He had anicteric sclera, clear breathing sounds, and regular heart beats. Direct tenderness in the RUQ and epigastrium was noticed without hepatosplenomegaly. Physical examination was otherwise unremarkable.

Initial laboratory tests showed white blood cell count of 22.3 × 10^9/L with 79% neutrophils, 7% lymphocytes, and 14% monocytes, C-reactive protein (CRP) of 20 mg/dL, aspartate aminotransferase of 95 IU/L, and alanine aminotransferase of 189 IU/L. Results of total bilirubin, alkaline phosphatase, prothrombin time, and fasting glucose were within the normal ranges. Initial chest and abdomen radiographs showed unremarkable findings. To find an intra-abdominal abscess, computed tomography (CT) was performed. The CT showed an ill-defined cluster of hypoattenuating lesions, consistent with multiple pyogenic liver abscesses (Fig. 1). Immediately after hospitalization to the pediatric ward, percutaneous catheter drainage was performed for 2 large lesions in segments 6 and 7, and drained pus was sent for culture. Intravenous ceftriaxone (50 mg/kg/day) and metronidazole (30 mg/kg/day) were administered as empirical antibiotics.

On day 4, *K. pneumoniae* was isolated from the drained pus. No bacteria were isolated from the initial blood culture. Serologic tests for *Entamoeba histolytica*, hepatotropic viruses (hepatides A, B, and C), and immunoglobulins G, A, and M were negative or within the normal ranges. On day 5, right pleural effusion was observed on a chest radiograph (Fig. 2), but no tube thoracostomy was performed given the absence of dyspnea. On day 7, ceftriaxone was replaced with ceftazidime.

Fig. 1. Computed tomography showing an ill-defined cluster of hypoattenuating lesions in hepatic segment 6 (5.0 cm, arrows), segment 7 (7.2 cm, arrowheads), and segment 4 (2.0 cm, not shown in this section). The clustered hypoattenuating lesions were consistent with multiple pyogenic liver abscesses.

Fig. 2. Right pleural effusion (arrows) on a chest radiograph.
(100 mg/kg/day) based on the sustained fever and the lowest minimum inhibitory concentration for *K. pneumoniae* among the antibiotics tested on the initially drained pus. On day 12, temperature, leukocytosis, and elevated levels of CRP and aminotransferases were normalized. On day 17, sizes of the abscesses decreased in segments 6 (2.7 cm), 7 (5.0 cm), and 4 (1.2 cm) on a follow-up CT. On day 18, we removed the catheters, and noted spontaneous resolution of the pleural effusion. After discharge on day 22, per os cefdinir was administered for 3 weeks. An abdominal ultrasound (US) performed upon completion of the antibiotic therapy showed an almost resolution of the PLA.

**2. Case 2**

A previously healthy 14-year-old boy visited the ED with RUQ and epigastric pain lasting for 2 days without antibiotic therapy. The initial temperature was 38.1°C. He showed anicteric sclera and direct tenderness in the RUQ and epigastrium without hepatosplenomegaly. Physical examination was otherwise unremarkable. Initial laboratory tests showed white blood cell count of 8.2 × 10⁹/L with 54% neutrophils, and CRP of 9.2 mg/dL. Aminotransferase levels were within the normal ranges. CT showed a 2.5 cm-sized peripherally enhancing, centrally hypoattenuating lesions, consistent with PLA (Fig. 3).

Immediately after hospitalization, US-guided percutaneous needle aspiration with a 20 G needle was performed, and aspirated 4 mL of brown-colored pus was sent for culture. Intravenous ceftriaxone and metronidazole were administered. A drainage catheter was not inserted due to the size of the US-confirmed lesion, which was smaller than that of the CT-confirmed lesion. On day 4, temperature and elevated level of CRP were normalized, and *K. pneumoniae* was isolated from the aspirated pus without a yield of the initial blood culture. Serologic tests for *E. histolytica* and hepatotropic viruses were negative. On day 15, a follow-up CT showed a 1.6 cm-sized residual abscess. After discharge on day 16 and administration of per os cefdinir for 4 weeks, the residual abscess disappeared on a follow-up US.

**Discussion**

For microbiological diagnosis of PLA, emergency physicians and pediatricians should perform blood or pus culture, *S. aureus* is the most common bacterial pathogen of PLA in children. However, a Taiwanese nationwide study on PLA shows *K. pneumoniae* as the most common bacterial pathogen. According to the studies on PLA performed in Asian population, *Klebsiella* species comprised 43%-66% of PLA cases, and was

---

*Fig. 3. Computed tomography showing a 2.5 cm-sized, hypoattenuating lesion (arrows) surrounded by an irregularly enhancing inner rim and a hypoattenuating outer rim in hepatic segment 6.*
typically in association with diabetes or of cryptogenic origin\textsuperscript{11-13}. Previously, *Escherichia coli* and anaerobes were known as common PLA pathogens in Korea, but recently, *K. pneumoniae* has become the most common pathogen\textsuperscript{4,5}. In this case report, predisposing factors for the Klebsiella infections are unknown due to the lack of risk factors for PLA.

PLA due to *K. pneumoniae* often leads to end-organ seeding and subsequent formation of extrahepatic abscesses. The extrahepatic abscesses have been most commonly reported in the eyes, and also have been observed in the lung, meninges, kidney, skin, brain, and bone\textsuperscript{14}. Instead of extrahepatic abscesses, we found the pleural effusion in patient 1. No tube thoracostomy was performed because the effusion was thought to be due to sympathetic stimulation of PLA\textsuperscript{15}.

Hsu et al.\textsuperscript{16} reported that fever and abdominal pain were the major manifestations of PLA. Other manifestations, such as diarrhea, vomiting, and weight loss are nonspecific. In this case report, patient 1 reported fever, RUQ pain, and vomiting. Although the sole symptom at presentation of patient 2 was RUQ pain, fever was found in the ED, so fever and abdominal pain could have been the clues to perform the CTs.

The laboratory findings in PLA are nonspecific. Leukocytosis, elevated levels of CRP and aminotransferases are commonly found\textsuperscript{17}. In this case report, patient 1 showed not only leukocytosis and elevated levels of CRP, but also elevated levels of aminotransferases. However, patient 2 showed only elevated levels of CRP. We speculated that he visited the ED earlier than did patient 1.

In emergency settings, imaging study is most important in diagnosis of PLA. Both US and CT provide visual identification, anatomical localization, and measuring size of PLA\textsuperscript{17}. In this case report, CT was used to find intra-abdominal abscesses so that we could make the expeditious diagnoses in the ED.

The treatment of choice is dual therapy with use of antibiotics and insertion of percutaneous catheter drainage\textsuperscript{18,19}. Initial combination of antibiotics may consist of anti-staphylococcal drugs (e.g., extended spectrum beta-lactams), anti-anaerobic and anti-amoebic drugs (e.g., metronidazole), and aminoglycosides or third generation cephalosporin for gram negative bacilli, such as *K. pneumoniae*\textsuperscript{20}. In case 1, after the CT diagnosis, we immediately performed percutaneous catheter drainage (in place for 18 days) in the 2 large lesions, and started empirical antibiotic therapy (ceftriaxone and metronidazole). In case 2, we performed an US-guided percutaneous needle aspiration. In both cases, after 6 weeks of antibiotic therapy, the initially noted PLAs showed the US-confirmed resolutions.

We report 2 cases of PLA due to *K. pneumoniae* in immunocompetent children diagnosed by CT. After a 6-week course of antibiotic therapy, resolution of the PLAs was noted without complications except the pleural effusion of patient 1. Despite the nonspecific nature of manifestations and laboratory findings, abdominal US or CT in the ED are strongly advised in children with a combination of fever, RUQ pain and abnormal laboratory findings, including leukocytosis, elevated levels of CRP and aminotransferases.

**Conflicts of interest**

No potential conflicts of interest relevant to this article were reported.

**Acknowledgements**

No funding source relevant to this article was reported.
References

1. Kliegman RM, Stanton BF, St. Geme JW, Schor NF. Nelson textbook of pediatrics. 20th ed. Philadelphia (PA): Elsevier; 2016. p. 1953-4.
2. Ferreira MA, Pereira FE, Musso C, Dettogni RV. Pyogenic liver abscess in children: some observations in the Espirito Santo State, Brazil. Arq Gastroenterol 1997;34:49-54.
3. Muorah M, Hinds R, Verma A, Yu D, Samyn M, Miel-Vergani G, et al. Liver abscesses in children: a single center experience in the developed world. J Pediatr Gastroenterol Nutr 2006;42:201-6.
4. Chung DR, Lee SS, Lee HR, Kim HB, Choi HJ, Eom JS, et al. Emerging invasive liver abscess caused by K1 serotype Klebsiella pneumoniae in Korea. J Infect 2007;54:578-83.
5. Chung DR, Lee HR, Lee SS, Kim SW, Chang HH, Jung SI, et al. Evidence for clonal dissemination of the serotype K1 Klebsiella pneumoniae strain causing invasive liver abscesses in Korea. J Clin Microbiol 2008;46:4061-3.
6. Chen YC, Lin CH, Chang SN, Shi ZY. Epidemiology and clinical outcome of pyogenic liver abscess: an analysis from the National Health Insurance Research Database of Taiwan, 2000-2011. J Microbiol Immunol Infect 2016;49:646-53.
7. Sharma A, Mukewar S, Mara KC, Dierkhising RA, Kamath PS, Cummins N. Epidemiologic factors, clinical presentation, causes, and outcomes of liver abscess: a 35-year Olmsted county study. Mayo Clin Proc Innov Qual Outcomes 2018;2:16-25.
8.Chan KS, Chen CM, Cheng KC, Hou CC, Lin HJ, Yu WL. Pyogenic liver abscess: a retrospective analysis of 107 patients during a 3-year period. Jpn J Infect Dis 2005;58:366-8.
9. Thomsen RW, Jepsen P, Sorensen HT. Diabetes mellitus and pyogenic liver abscess: risk and prognosis. Clin Infect Dis 2007;44:1194-201.
10. Tsai FC, Huang YT, Chang LY, Wang JT. Pyogenic liver abscess as endemic disease, Taiwan. Emerg Infect Dis 2008;14:1592-600.
11. Lee KT, Wong SR, Sheen PC. Pyogenic liver abscess: an audit of 10 years’ experience and analysis of risk factors. Dig Surg 2001;18:459-65.
12. Tan EY, Lee CW, Look Chee Meng M. Spontaneous pneumoperitoneum resulting from the rupture of a gas-forming pyogenic liver abscess. ANZ J Surg 2005;75:251-2.
13. Foo NP, Chen KT, Lin HJ, Guo HR. Characteristics of pyogenic liver abscess patients with and without diabetes mellitus. Am J Gastroenterol 2010;105:328-35.
14. Cheng DL, Liu YC, Yen MY, Liu CY, Wang RS. Septic metastatic lesions of pyogenic liver abscess. Their association with Klebsiella pneumoniae bacteremia in diabetic patients. Arch Intern Med 1991;151:1557-9.
15. Light RW. Pleural diseases. 4th ed. Baltimore (MD): Lippincott Williams & Wilkins; 2001. p. 237-45.
16. Hsu YL, Lin HC, Yen TY, Hsieh TH, Wei HM, Hwang KP. Pyogenic liver abscess among children in a medical center in Central Taiwan. J Microbiol Immunol Infect 2015;48:302-5.
17. Israeli R, Jule JE, Hom J. Pediatric pyogenic liver abscess. Pediatr Emerg Care 2009;25:107-8.
18. Byun JL, Bae SH, Park SW. A case of pyogenic liver abscess in a 10-year-old girl. Korean J Pediatr 2010;53:666-8.
19. Johannsen EC, Sifri CD, Madoff LC. Pyogenic liver abscesses. Infect Dis Clin North Am 2000;14:547-63.
20. Sharma MP, Kumar A. Liver abscess in children. Indian J Pediatr 2006;73:813-7.