To the Editor,

*Klebsiella pneumoniae* is the major pathogen of community-acquired infections in Taiwan.1 *K. pneumoniae* liver abscess (KPLA) has been reported with increasing frequency in East Asian countries in the past 3 decades, especially in Taiwan.1,2,3 Gas-forming pyogenic liver abscess (GFPLA) is uncommon and associated with a high fatality rate. The literature suggested that mixed acid fermentation of glucose was the mechanism of gas formation.4 *K. pneumoniae* is the most common pathogen of GFPLA in Taiwan.4 GFPLA due to *K. pneumoniae* usually occur in patients with underlying diabetes mellitus (DM), especially among those with poor glycemic control.2,4

We describe a case without DM diagnosed as GFPLA due to *K. pneumoniae* at Taipei Veterans General Hospital in Taiwan. In addition, the presence of common virulence factors of *K. pneumoniae* strain was examined.

A 72-year-old gentleman with a medical history of hypertension was admitted to the hospital because of fever for 4 days. Initially, tachycardia with leukocytosis (17,700/μL), elevated C-reactive protein (35.6 mg/dL), high aspartate aminotransferase (206 IU/L), high alanine aminotransferase (248 IU/L), and high alkaline phosphatase (300 IU/L) were noted. The patient reported no history of DM. His fasting glucose 3 months ago was 105 mg/dL, and his hemoglobin A1C level (checked at this admission) was 6.2%. The computed tomography image (Fig. 1) showed a mottled gas pattern with low density about 8 cm at the left lobe of the liver, which revealed a gas-forming liver abscess. He was treated empirically with doripenem, and the abscess was drained percutaneously. Septic shock ensued and he received intensive care in the intensive care unit. Cultures from blood and liver abscess both yielded *K. pneumoniae*, which was susceptible to a number of classes of antibiotics, except for an intrinsic resistance to ampicillin by the VITEK 2 system (bioMérieux, Marcy l’Etoile, France). The patient received ceftriaxone and then cefotaxime because of liver function impairment as the definitive antimicrobial therapy. An abdominal ultrasonography conducted after the completion of a 6-week antibiotic course demonstrated resolution of his liver abscess.

The *K. pneumoniae* blood isolate was hypermucoviscous, as shown by the formation of a mucoviscous strings when a loop was passed through a colony. The capsular genotype was K1, and it belonged to ST23 as determined by the *K. pneumoniae* MLST scheme. The isolate was also positive for *rmpA* and *rmpA2*. In diabetic patients with KPLA, gas-forming abscess was not identified in patients with controlled glycemia (hemoglobin A1c <7%).4 The capsular type of *K. pneumoniae* for GFPLA has never been determined in the literature. To the best of our knowledge, this
is the first case of GFPLA due to capsular type K1 K. pneumoniae in nondiabetic patients.

DM is a well-known risk factor for KPLA, and glycemic control in diabetic patients played an essential role in the clinical characteristics of KPLA. However, we found more KPLA in non-DM patients in our hospital recently. The current case further demonstrated that the virulent K. pneumoniae clone ST23 had the ability to cause GFPLA in a nondiabetic patient. As an endemic disease in Taiwan, although glycemic level plays an important role in the pathogenesis of KPLA, further study focusing on the characteristics of non-DM patients with KPLA is necessary.

Conflicts of interest

All authors declare no conflicts of interest.

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References


