determined using both assays, 34 (24%) had test results with discrepancies of >0.5 log10 copies/mL. Similarly, Oliver et al [6] reported that 7 (3.7%) of 187 patients had TaqMan assay results that were at least 0.5 log10 copies/mL lower than their Amplicor assay results and that 5 (3.2%) of 187 patients had TaqMan assay results that were at least 0.5 log10 copies/mL higher than their Amplicor assay results. In addition, 12 (6.4%) of 187 patients with a detectable TaqMan assay result had an undetectable Amplicor assay result. Similarly, Manavi [7] reported that 16 (14%) of 113 patients had a detectable TaqMan assay result and an undetectable Amplicor assay result. Finally, Lima et al [8] reported that the TaqMan assay resulted in a nearly 2-fold increase in the number of patients (from 3.6% to 6.9%) with a plasma HIV-1 RNA level of >50 copies/mL (but <250 copies/mL) after being consistently suppressed to levels of <50 copies/mL as assessed by the Amplicor assay (P < .01).

These studies consistently show that use of the TaqMan assay results in detectable plasma HIV-1 RNA levels above the critical clinical threshold of 50 copies/mL for a substantial proportion of individuals whose virus was previously considered consistently suppressed to levels of <50 copies/mL using the Amplicor assay. This increased frequency of detectable plasma HIV-1 RNA as a result of using the TaqMan assay has important implications for the monitoring of antiretroviral therapy, including the need for additional viral-load testing and medical visits, which, in turn, puts an additional stress on patients and health care providers. Furthermore, these differences may prove crucial in the outcomes of clinical trials, which typically have a primary efficacy endpoint of achieving plasma HIV-1 RNA levels of <50 copies/mL. The discrepancies outlined above are partially a result of the significant widening of the coefficients of variation of these assays at the lower limits of the quantitative range, with the limit of detection below the lower quantitative threshold for the assays. Nevertheless, the clinical consequences for patients and health care providers who are informed that a regimen is no longer suppressing HIV replication can be substantial. It is important to appreciate that a change in assay can be misleading as it can result in the appearance of virological failure that is not attributable to patient adherence or regimen inadequacy.

The currently available data cannot shed light on the long-term prognostic significance of patients whose more recent TaqMan assay results (ie, low but detectable plasma HIV-1 RNA levels of >50 copies/mL) were at odds with their less recent Amplicor assay results (ie, HIV-1 RNA levels of <50 copies/mL). HIV caregivers should be knowledgeable about which assay is being used to determine their patients' plasma HIV-1 RNA levels, and they should be aware of the role that assay performance can play at the lower limit of assay sensitivity, so that appropriate clinical judgement can be brought to bear in interpreting the results.

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Repeated Bacteremia with Subsequent Septic Arthritis Caused by Klebsiella pneumoniae Capsular Serotype K57 in a Patient with Diabetes

To the Editor—Invasive infections caused by Klebsiella pneumoniae in patients with diabetes are frequently caused by capsular serotypes K1 and K2, which
been hospitalized 4 times because of receive any further treatment. She had carcinoma in September 2005 and did not received a diagnosis of hepatocellular carcinoma at least as early as April 2005. She had virus–related liver cirrhosis (Child-Pugh of type 2 diabetes mellitus and hepatitis B pain over the right knee. She had a history week history of fever, abdominal pain, and to the emergency department with a 1-

Figure 1. Pulsed-field gel electrophoresis profiles of XbaI-digested genomic DNA from 7 Klebsiella pneumoniae isolates. Lanes 1–4 show profiles for isolates A, B, C, and D, respectively, from the patient with septic arthritis and repeated bacteremia due to K. pneumoniae; lines 5–7 show profiles for isolates E, F, and G, respectively, of K. pneumoniae from 3 other patients that were used as control strains. Lane M, bacteriophage λ DNA concatemers (GibcoBRL). bp, base pairs.

are the most virulent of the known serotypes [1, 2]. Bacteremia and septic arthritis due to K. pneumoniae capsular serotype K57 has never, to our knowledge, been reported [1, 3]. We here describe a case of repeated bacteremia and septic arthritis caused by K. pneumoniae serotype K57 in a diabetic and cirrhotic patient with hepaticellular carcinoma.

A 62-year-old female patient presented to the emergency department with a 1-week history of fever, abdominal pain, and pain over the right knee. She had a history of type 2 diabetes mellitus and hepatitis B virus–related liver cirrhosis (Child-Pugh C) at least as early as April 2005. She had received a diagnosis of hepatocellular carcinoma in September 2005 and did not receive any further treatment. She had been hospitalized 4 times because of K. pneumoniae bacteremia, at 18 months, 17 months (isolate A), 2 months (isolate B), and 1 month (isolate C) before this hospital admission. The first episode of bacteremia was associated with community-acquired pneumonia and had been treated with ceftriaxone for 14 days. The latter 3 episodes were primary bacteremia treated with ceftriaxone followed by amoxicillin-clavulanate (for 14 days), ceftazidime followed by oral cefuroxime (for 20 days), and flomoxef followed by oral cefuroxime (for 16 days).

At presentation, the patient’s temperature was 37.8°C, her pulse rate was 91 beats/min, her respiration rate was 18 breaths/min, and her blood pressure was 111/59 mmHg. Amoxicillin-clavulanate (1 g administered twice daily) was given orally initially. On physical examination, the patient was icteric. Her right knee was erythematous, tender, and swollen, with local heat. Laboratory examinations disclosed a hemoglobin level of 11 g/dL, a white blood cell (WBC) count of 12.4 × 10^9 cells/L (10.9 × 10^9 neutrophils/L), a platelet count of 64 × 10^10 platelets/L, a creatinine level of 1 mg/dL, a total bilirubin of 2.97 mg/dL, a C-reactive protein level of 12.71 mg/dL, and a glycosylated hemoglobin level of 9.7%. Levels of serum aspartate aminotransferase, alanine aminotransferase, amylase, and ammonia were normal. Aspiration of the right knee yielded 10 mL of turbid and yellowish synovial fluid with a WBC count of 84,336 × 10^6 cells/L (92% neutrophils). Abdominal ultrasonography showed cholecystolithiasis and minimal ascites and the absence of liver abscesses. Findings of triple-phase bone scan were compatible with septic arthritis.

Culture of the aspirated synovial fluid grew K. pneumoniae (isolate D); however, 2 sets of blood cultures (Bactec 9240; Becton Dickinson) were negative for the organism. Ceftriaxone therapy (2 g every 12 h) was initiated on the third hospital day. The patient was treated with repeated arthrocentesis and intravenous ceftriaxone for 4 weeks and responded well.

Each of the 4 K. pneumoniae isolates were identified as capsular serotype K57 by polymerase chain reaction (PCR)–restriction fragment-length polymorphism and PCR-sequencing analysis of the capsular polysaccharide synthesis regions as described elsewhere [1]. Antimicrobial susceptibility testing with use of Phoenix ID/AST system (NMIC/ID-4; Becton Dickinson) for the 4 isolates (isolates A, B, C, and D) revealed identical susceptibility profiles. All isolates were resistant to amoxicillin-clavulanate (minimum inhibitory concentration [MIC], >16 μg/mL) and susceptible to ceftriaxone (MIC, ≤1 μg/mL).

Randomly amplified polymorphic DNA (RAPD) analysis of the 4 K. pneumoniae isolates from the patient (isolates A, B, C, and D) and 3 K. pneumoniae isolates (isolates E, F, and G) recovered from cultures of blood samples obtained from 3 epidemiologically unrelated patients (obtained in 2005, 2006, and 2007, respectively) were also performed [4]. The random primers AP4 (5′-TCACGTATGCA-3′), H1WL74 (5′-ACGTATCTGC-3′), and R108 (5′-GTATTGCCCT-3′) were used. Pulsed-field gel electrophoresis typing of XbaI-digested genomic DNA was performed as described elsewhere [5]. Iden-
tactical RAPD patterns and pulsotypes (Figure 1) were found in the 4 isolates (isolates A, B, C, and D). RAPD patterns and pulsotypes of isolates E, F, and G were different from those of A, B, C, and D.

We report a case with repeated bacteremia and septic arthritis due to K. pneumoniae serotype K57 in a patient with multiple underlying medical conditions (diabetic mellitus, liver cirrhosis, and hepatocellular carcinoma). To the best of our knowledge, 19 cases of septic arthritis due to K. pneumoniae have been reported in the English-language literature [1, 6–9]. Among 13 patients with Klebsiella arthritis reviewed by Schelenz et al [3], 4 had diabetes mellitus, 1 had liver cirrhosis, and 1 had malignancy. Findings from a recent study indicate that diabetes mellitus is strongly correlated with recurrent K. pneumoniae infections [10].

The identical pulsotypes and RAPD patterns of the 4 K. pneumoniae isolates recovered within an 18-month period from our patient indicated that the unidentified and noneliminated primary foci were responsible for repeated bacteremia, which resulted in septic arthritis due to the same strain of K. pneumoniae.

A previous study has concluded that patients with pyogenic liver abscess caused by non-K1 K. pneumoniae are considered to be more associated with diabetes mellitus, compared with those with infections caused by serotype K1 [1]. Impaired phagocytosis of capsular serotypes K1 or K2 K. pneumoniae is found in diabetic patients with poor glycemic control [2]. Little is known about patients infected with serotype K57 K. pneumoniae. The impaired phagocytosis of non-K1 K. pneumoniae strains in patients with poor diabetic control (glycosylated hemoglobin level >9.0%) might partly contribute to the repeated bacteremia that occurred, despite adequate antimicrobial treatment, in our patient.

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