test [Quidel]). Quality-improvement initiatives identified inappropriate specimen procurement, incorrect specimen containment, or delayed specimen shipment for 4.8% of the nasopharyngeal specimens obtained. A county-wide educational program on proper specimen procurement, transport, and processing occurred from 1 April through 31 August 2004. After the educational program, 4417 patients had at least 1 specimen (range, 1–6 specimens per patient) submitted for H5N1 testing. There was a notable reduction in suboptimal specimen collections to 2.4% (P = .02). Thirteen (0.3%) of 4417 patients had H5N1 infection confirmed by RT-PCR, real-time RT-PCR, and/or viral culture results. Ten (77%) of these 13 patients had nasopharyngeal rapid testing, and 3 (30%) of these 10 patients had nasopharyngeal rapid test results positive for H5N1. Of note, 1 patient with H5N1 infection received neuraminidase inhibitor ≥48 h before specimen collection and had negative nasopharyngeal rapid test results.

Our findings have some important implications. Although physicians tend to submit multiple specimens from each index patient, and although suboptimal specimen collection and processing were identified less commonly after the educational program, we found no difference in the diagnostic yield of the rapid test. With the increase in neuraminidase inhibitor supply in Thailand, physicians tend to prescribe this medication to the index patient long before specimen collection. Because rapid diagnosis for H5N1 infection can be difficult [3], we emphasize the importance of treating physicians obtaining multiple adequate, deep specimens from patients before the administration of antiviral medication to the index patient.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

Rapidly Fatal Gas-Forming Pyogenic Psoas Abscess Caused by Klebsiella pneumoniae

To the Editor—Bilateral gas-forming pyogenic psoas abscesses caused by Klebsiella pneumoniae have rarely been reported in the English-language literature [1–3]. We describe a 78-year-old man who had a 10-year history of hypertension and diabetes mellitus controlled with antihypertensive and oral hypoglycemic agents.

Eight weeks before this visit, the patient had presented to the emergency department with high fever and rigors. Abdominal CT revealed a liver abscess (size, $3 \times 4$ cm) over the right lobe. No metastatic lesions, such as endophthalmitis, or involvement of psoas or spine was found during the hospitalization (figure 1A). Repeated echo-guided aspiration was required before resolution of the abscess. Intravenous ceftriaxone (1 g every 12 h) was administered for 2 weeks, followed by oral cefixime (200 mg every 12 h) for 4 weeks. Cultures of blood samples (isolate A) and liver aspirate specimens (isolate B) both yielded K. pneumoniae with the hypermucoviscosity phenotype [1]; the isolate was determined to be susceptible to cefazolin, cefmetazole, and ceftriaxone by the standard disk diffusion method.

Two weeks after completion of the antibiotic regimen for treatment of the liver abscess, the patient presented to the emergency department again with fever (temperature, $\approx 38.6^\circ\text{C}$) and rigors with a 1-day duration. There were no other characteristic symptoms, such as dysuria, abdominal pain, or flank pain. Laboratory studies revealed a WBC count of 9040 cells/mm$^3$ (29% bands and 57% neutrophils) and a blood sugar level of 181 mg/dL. Empirical ceftriaxone treatment (1 g every 12 h) was initiated because of the

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recent history of *K. pneumoniae* liver abscess. Abdominal CT was performed 4 h after the patient’s arrival and revealed an extensive gas-forming pyogenic psoas abscess, which extended from the bilateral psoas muscles to the pelvis, with involvement of the third and the fourth lumbar spines (figure 1B). The previously treated liver abscess was confirmed to have resolved completely. However, refractory shock and severe acidosis (serum lactate level, 11.97 mmol/L) developed rapidly. The patient died 10 h after arrival. Two consecutive blood cultures both yielded *K. pneumoniae* (isolate C) with antibiotype (isolate A) and hypermucoviscosity phenotype; the isolates were similar to the previous isolates.

The presence of the mucoviscosity-associated gene A (*magA*), 22-kb chromosomal region (*alls*), and *rmpA* genes in the 3 *K. pneumoniae* isolates was determined by PCR using the primers described elsewhere [4–7]. All 3 *K. pneumoniae* isolates were positive for *rmpA* gene but negative for *magA* gene and the 22-kb chromosomal region. Pulsotypes of these 3 *K. pneumoniae* isolates and 4 epidemiologically unrelated *K. pneumoniae* isolates recovered from blood samples (controls) were determined by PFGE using XbaI restriction enzyme [8]. The 3 isolates (isolates A–C) had identical pulsortypes that were different from those of the 3 control isolates (figure 2).

Bilateral psoas abscess with gas formation caused by *K. pneumoniae* that occurs after adequate treatment of liver abscess has, to our knowledge, never been reported in the English-language literature. Of the reported cases of psoas abscess due to *K. pneumoniae*, none of the lesions were gas forming, and one of the patients had concomitant liver abscess [1–3]. The pulsortype of isolates recovered from the bilateral gas-forming psoas abscess in our patient was identical to that of the isolates from his recent liver abscess. This indicated that the infection had relapsed, even though previous symptoms had resolved during a 6-week course of effective antibiotic treatment.

The *K. pneumoniae* isolates recovered from this patient exhibited hypermucoviscosity phenotype and were *rmpA* positive and *magA* negative. The presence of *rmpA* and/or *magA* genes in *K. pneumoniae* is associated with the hypermucoviscosity phenotype and purulent tissue infection [4, 5, 7]. The 22-kb chromosomal region in *K. pneumoniae* isolates was also reported to be significantly associated with liver abscess [6]. Although gas-forming psoas abscess due to *K. pneumoniae* has a high mortality rate [7], the presence of *rmpA*, the 22-kb chromosomal region, or other virulence factors may have also contributed to the rapidly fatal outcome in this immunocompromised patient.

In conclusion, the presentation of fever in a diabetic patient with a recent history of *K. pneumoniae* liver abscess should raise suspicion about a recurrent episode and/or the presence of septic metastatic lesions. *K. pneumoniae* has the potential to cause bilateral gas-forming psoas abscess, especially in diabetic patients. Prompt diagnosis, aggressive antibiotic treatment, and immediate surgical intervention are crucial for management of this life-threatening condition.

**Acknowledgments**

**Potential conflicts of interest.** All authors: no conflicts.

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**Figure 2.** PFGE profiles of XbaI-digested genomic DNA from 7 *Klebsiella pneumoniae* isolates. Lanes 1–4, profiles of 4 epidemiologically unrelated *K. pneumoniae* isolates recovered from blood samples and used as control strains; lanes 4–6, profiles for isolates A–C, respectively, which were recovered from the patient described in this report; lane M, bacteriophage λ DNA concatemers (GibcoBRL).

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Emergence of Fluoroquinolone Only–Resistant Escherichia coli in a Community Hospital

To the Editor—We report the emergence in a community hospital of Escherichia coli isolates, obtained from urine samples, that are resistant only to fluoroquinolones and were identified retrospectively beginning in 2001. In 2005, 2 healthy, immunocompetent patients received diagnoses of uncomplicated urinary tract infection and were treated empirically with levofloxacin pending culture results. Neither of the patients responded to treatment with levofloxacin. Identification and susceptibility tests performed with the Vitek (bioMérieux) system on samples obtained before the administration of antibiotics identified E. coli that were resistant to levofloxacin and ciprofloxacin but were susceptible to all other antibiotics tested, including trimethoprim-sulfamethoxazole. Each patient’s therapy was promptly switched to an antibiotic to which the organism was susceptible, with subsequent resolution of symptoms. Because E. coli isolates that are resistant only to fluoroquinolone antibiotics—specifically, levofloxacin and ciprofloxacin—are considered to be uncommon [1], an effort was made to retrospectively determine the frequency and prevalence of fluoroquinolone only–resistant E. coli (FORE) identified in our laboratory using the Vitek Data Trac software program (bioMérieux). Including only unique patient isolates, the percentage of isolates reported in our laboratory that were identified as FORE for each year from 1998 to 2006 was 0%, 0%, 0.10% (1 isolate), 0.21% (2 isolates), 0%, 0.27% (3 isolates), 0.88% (10 isolates), and 1.36% (16 isolates), respectively. Because none of the isolates with the FORE phenotype obtained before 2005 were available for testing, we sent the first 6 isolates recovered after the trend was identified to an external reference laboratory for confirmatory susceptibility testing. This laboratory confirmed our initial results using the same method (Vitek) and using the microbroth dilution method. PFGE testing of 11 of the isolates obtained in 2006 by the Massachusetts Department of Public Health revealed that the banding patterns for these 11 isolates were different, suggesting that clonal spread of this phenotype is not occurring at present and that this is a heterogeneous phenomenon.

When reported, resistance to fluoroquinolones in E. coli is usually observed in conjunction with resistance to other antibiotic classes [1–3]. Therefore, our observation of the emergence and increased frequency of FORE is unusual and somewhat perplexing. From an epidemiological standpoint, our concern is that we have identified the emergence of an E. coli phenotype that, once established, may persist and increase in frequency, particularly in the face of selection pressure. Of note, as a contrast to our findings, data from large antibiotic-resistance surveillance studies in the United States suggest that the frequency of FORE is not increasing [Tracking Resistance in the United States Today study from Ortho-McNeil Pharmaceuticals, unpublished data].

From a clinical standpoint, our concern is that physicians currently assume, as they should, that a fluoroquinolone has greater empirical value than older agents, such as ampicillin, for treating uncomplicated E. coli infection. Empirically, it is counterintuitive to think that a fluoroquinolone-resistant E. coli isolate would be susceptible to all other antibiotics tested, including ampicillin and cefazolin, which are not typically considered to be first- or second-line agents against gram-negative infection. Our experiences suggest that clinicians need to become aware of the FORE phenotype in their area, especially in situations with little room for therapeutic error.

Acknowledgments
Potential conflicts of interest. All authors: no conflicts.

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Clinical Infectious Diseases 2007;44:1255–5
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