parts of the world [1]. And we appreciate that Yu and Chang provided some unpublished data to strengthen our earlier hypothesis that some K. pneumoniae strains in Taiwan may have characteristics that influence their invasiveness. Many investigators have tried to determine why K. pneumoniae-associated liver abscess is common in Taiwan but not in other countries. K. pneumoniae-associated liver abscess in Taiwan is not caused by a clonal spread strain [13, 14], and the virulence was possibly related to a different serotype or magA-positive virulent gene [14, 15].

The incidence of K. pneumoniae as the causative agent of liver abscess in Taiwan increased from 30% in the 1980s to 88% in the 1990s [16, 17]. As described in the discussion section of our article, the proportion of K. pneumoniae isolates in cases of lung abscess in Taiwan, as diagnosed by examination of transthoracic aspirate specimens, increased from 5% in the late 1980s to 21% in the late 1990s [2]. Yu and Chang [1] mentioned that they found scattered cases of invasive infection caused by virulent strains worldwide (including in the United States) in their unpublished observations. Two studies of liver abscess from the United States recently found that K. pneumoniae-associated liver abscess is an emerging disease [1, 18, 19]. All of these data suggest that the increasing prevalence of invasive K. pneumoniae infection may not only be happening in Taiwan, but it may also occur in other parts of world in upcoming years. This finding deserves attention, because dissemination of these highly virulent K. pneumoniae strains from Taiwan to other countries is not impossible.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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References


Fungemia Secondary to Gastrointestinal Mucor indicus Infection

Sir—We describe the case of a 34-year-old man who presented to the emergency department with a 1-h history of severe abdominal pain, diarrhea, and fever. Three hours before, the patient had eaten a partially reheated meal that had been unrefrigerated for 24 h. His past medical history included intravenous amphotericin use up to 3 years before presentation. On examination, the patient was tachycardic and hypotensive. There was severe abdominal tenderness.

The patient received broad-spectrum intravenous antibiotics, intravenous fluids, and inotropic support. In 12 h, acute renal failure and acute respiratory distress syndrome had developed. A CT of the abdomen revealed thickening of the wall of the cecum, stranding of the surrounding fat, and free fluid in the right paracolic gutter. Three sets of blood samples for culture were obtained during the first 3 days after admission. All grew a branching, filamentous fungus that was identified by both phenotypic and genotypic methods as Mucor indicus. Feces culture grew the same organism. No organisms were cultured from sputum. Tranosophageal echocardiography revealed normal heart valves.

Treatment commenced with intrave-
nous amphotericin B lipid complex (5 mg/kg per day), which was then switched to liposomal amphotericin B (5 mg/kg per day). There was no surgical intervention. For the first 7 days of hospitalization, the patient remained unwell in the intensive care department, with ongoing fever, profuse diarrhea, and abdominal tenderness. There was then a steady recovery. After 3 weeks, the patient had stabilized and had no gastrointestinal symptoms. Amphotericin B lipid preparations were administered for a total of 6 weeks. The patient remained healthy at follow-up 3 months later.

Gastrointestinal mucormycosis is an uncommon disease. It has most often been described in patients with diabetes and malnutrition, infants with low birth weight, patients receiving peritoneal dialysis, and patients with solid organ transplants [1, 2]. It occurs most commonly in the stomach (60% of cases) and the colon (30% of cases), with half of the colonic cases occurring in the cecum and ascending colon [3]. The infection is thought to be acquired through direct ingestion of the pathogen [4]. The case presented here illustrates the rapid clinical onset and rapid clinical deterioration with which gastrointestinal mucormycosis often presents.

*Mucor indicus* (previously known as *Mucor rouxii*) has been reported to cause gastrointestinal mucormycosis in 40% of reported cases. Gastrointestinal mucormycosis is an uncommon disease. It has most often been described in patients with diabetes and malnutrition, infants with low birth weight, patients receiving peritoneal dialysis, and patients with solid organ transplants [1, 2]. It occurs most commonly in the stomach (60% of cases) and the colon (30% of cases), with half of the colonic cases occurring in the cecum and ascending colon [3]. The infection is thought to be acquired through direct ingestion of the pathogen [4]. The case presented here illustrates the rapid clinical onset and rapid clinical deterioration with which gastrointestinal mucormycosis often presents.

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