second patient were within the same subgroup, but similarity in nucleotide sequences was only 82.5%. This result suggests that this patient was infected with 2 different virus strains of HRV-A and a strain of HRV-C.

Comparison of the HRV-A strains with the HRV-C strain showed that they belonged to different subgroups and had low similarity for nucleotide sequences. The second patient had 3 distinct rhinovirus infections over 3 months, and each was associated with illness requiring hospitalization. Both patients had underlying diseases, reactive airway diseases, and repeated episodes of RTI that may have rendered them vulnerable to re-infection, compromising their immune responses.

Complete coding sequences of HRV-A and HRV-C have been determined (4,7). However, little is known about their involvement in the pathogenesis of recurrent wheezing in young children. According to recent reports, HRV-C has been detected in hospitalized children with lower RTI in the People’s Republic of China (5). Possible association of novel infection with HRV and exacerbation of asthma in children has also been reported (6). We report HRV-A and HRV-C co-infections in conjunction with other respiratory viruses, such as RSV, as a potential cause of recurrent wheezing in infants with acute lower RTIs. Co-infections with HRV-A and HRV-C may contribute to increased virulence and subsequent pathogenesis of other respiratory viruses. Additional studies will be required to further explore the clinical role of novel HRVs.

Acknowledgment

We thank P. Hirsch for reviewing the manuscript.

This study was supported by the Higher Commission of Education, Ministry of Education, Thailand, and The Center of Excellence Research Fund, Chulalongkorn University.

Piyada Linsuwanon, Sunchai Payungporn, Rujijat Samransamrakjit, Apiradee Theamboonlers, and Yong Poovorawan

Author affiliation: Chulalongkorn University, Bangkok, Thailand

DOI: 10.3201/eid1506.081558

References


Address for correspondence: Yong Poovorawan, Center of Excellence in Clinical Virology, Department of Pediatrics, Faculty of Medicine, Chulalongkorn University and Hospital, Rama IV Rd, Patumwan, Bangkok 10330, Thailand; email: yong.p@chula.ac.th

Extensively Drug-Resistant Acinetobacter baumannii

To the Editor: In the 1990s, patients infected with vancomycin-resistant Enterococcus faecium were successfully treated with new antimicrobial drugs. However, it is unlikely that new antimicrobial drugs will be available in the near future to treat patients infected with gram-negative pathogens such as Acinetobacter baumannii (1). No new antimicrobial drugs active against this organism are currently in clinical trials (www.clinicaltrials.gov). We report a patient infected with A. baumannii that lacked susceptibility to all commercially available antimicrobial drugs.

The patient, a 55-year-old woman, had a prolonged stay in an intensive care unit at the University of Pittsburgh Medical Center (Pittsburgh, PA, USA) after undergoing lung transplantation. In the tenth postoperative week, ventilator-associated pneumonia developed, which was caused by A. baumannii that lacked susceptibil-
ity to all antimicrobial drugs tested except colistin (MIC 0.5 µg/mL). Therapy with colistin and tigecycline was begun. Colistin was administered intravenously and by inhalation. Although the pneumonia showed radiographic response to the antimicrobial drug therapy, *A. baumannii* continued to be isolated from respiratory secretions on numerous occasions. Despite another course of therapy with colistin and ceftazidime, the patient never recovered from respiratory failure. She eventually died of sepsis caused by vancomycin-resistant *E. faecium*. An *A. baumannii* isolate obtained just before she died lacked susceptibility to all commercially available antimicrobial drugs (Table).

Multidrug-resistant *A. baumannii* has emerged as a substantial problem worldwide (2). Such strains are typically resistant to all β-lactams and fluoroquinolones and require salvage therapy with colistin, amikacin, or tigecycline. Unfortunately, notably high-level resistance to colistin and amikacin was found in the isolate we have described (Table). Tigecycline, a newly available glycylcycline antimicrobial drug, showed intermediate susceptibility. No randomized trials have been performed to specifically evaluate combination antimicrobial drug therapy for treatment of infection with *A. baumannii*.

Considerable media attention has been paid to extensively drug-resistant (XDR) strains of *Mycobacterium tuberculosis* (3). Infections with XDR strains are extremely difficult to treat and pose considerable infection control issues. We recently proposed that gram-negative bacilli lacking susceptibility to all commercially available antimicrobial drugs also be referred to as XDR because no therapeutic options are available (4).

Numerous outbreaks of *A. baumannii* infection have been reported worldwide (5). Unfortunately, multidrug-resistant *A. baumannii* strains have become endemic in some institutions. Experimental and clinical isolates lacking susceptibility to colistin, often considered the drug of last resort, are increasingly being reported (6–8). Therefore, we alert healthcare workers to the need for stringent care in adhering to infection control precautions when caring for patients infected with XDR *A. baumannii*. Use of contact isolation precautions, enhanced environmental cleaning, removal of sources of infection from the hospital environment, and prudent use of antimicrobial drugs can contribute to control of such outbreaks (5). Fortunately, no spread of the XDR strain affecting this patient occurred. A crisis is looming should XDR *A. baumannii* become established pathogens in hospitals.

Y.D. was supported by National Institutes of Health (NIH) training grant T32AI007333, and D.L.P. was supported in part by NIH research grant R01AI070896.

Yohei Doi, Shahid Husain, Brian A. Potoski, Kenneth R. McCurry, and David L. Paterson

Author affiliations: University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA (Y. Doi, B.A. Potoski, D.L. Paterson); University of Toronto, Toronto, Ontario, Canada (S. Husain); Toronto General Hospital, Toronto (S. Husain); Cleveland Clinic, Cleveland, Ohio, USA (K.R. McCurry); University of Queensland Centre for Clinical Research, Brisbane, Queensland, Australia (D.L. Paterson); and Royal Brisbane and Women’s Hospital, Brisbane (D.L. Paterson)

DOI: 10.3201/eid1506.081006

References


---

Table. MICs and antimicrobial drug susceptibility for an extensively drug-resistant strain of *Acinetobacter baumannii*.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>MIC, µg/mL</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbapenems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imipenem</td>
<td>&gt;32</td>
<td>Resistant</td>
</tr>
<tr>
<td>Meropenem</td>
<td>&gt;32</td>
<td>Resistant</td>
</tr>
<tr>
<td>Penicillins</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ampicillin/sulbactam</td>
<td>32</td>
<td>Resistant</td>
</tr>
<tr>
<td>Piperacillin/tazobactam</td>
<td>&gt;256</td>
<td>Resistant</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>48</td>
<td>Resistant</td>
</tr>
<tr>
<td>Cefepime</td>
<td>16</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gentamicin</td>
<td>&gt;256</td>
<td>Resistant</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>&gt;256</td>
<td>Resistant</td>
</tr>
<tr>
<td>Amikacin</td>
<td>&gt;256</td>
<td>Resistant</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>&gt;32</td>
<td>Resistant</td>
</tr>
<tr>
<td>Tigecycline</td>
<td>2</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Colistin</td>
<td>&gt;1,024</td>
<td>Resistant</td>
</tr>
</tbody>
</table>

*Susceptibility testing was performed by using the Etest (AB Biodisk, Solna, Sweden), except for colistin, for which the standard agar dilution method was used. Interpretation was according to breakpoints provided by the Clinical and Laboratory Standards Institute (CLSI; Wayne, PA, USA). No tigecycline breakpoints for *Acinetobacter* spp. are provided by the CLSI, the European Committee on Antimicrobial Susceptibility Testing (Basel, Switzerland), or the US Food and Drug Administration (Silver Spring, MD, USA). Breakpoints of the British Society for Antimicrobial Chemotherapy (Birmingham, UK) are indicated for tigecycline.*

Address for correspondence: Yohei Doi, University of Pittsburgh Medical Center, S829 Scaife Hall, 3550 Terrace St, Pittsburgh, PA 15261, USA; email: yod4@pitt.edu

**Cryptosporidium Pig Genotype II in Immunocompetent Man**

To the Editor: Protozoan parasites from the genus Cryptosporidium have been described as a cause of diarrheal disease in immunodeficient and immunocompetent humans worldwide. Although *C. hominis* and *C. parvum* (cattle genotype) cause most infections, humans can be infected by several other *Cryptosporidium* species or genotypes: *C. meleagridis*; *C. felis*; *C. canis*; *C. suis*; *C. muris*; *C. andersoni*; *C. hominis* monkey genotype; *C. parvum* (mouse genotype); and *Cryptosporidium* rabbit genotype, deer genotype, skunk genotype, horse genotype, and chipmunk genotype I (1–4). Wild and domestic animals are sources of infection for humans (and other animals) and important contributors to contamination of food and drinking water; many nonhuman *Cryptosporidium* species or genotypes are detected in untreated water (5). We examined the diversity of *Cryptosporidium* spp. in immunocompetent persons in South Bohemia in the Czech Republic.

Diarrheal fecal samples (n = 457) from 203 anonymous immunocompetent patients ≤ 69 years of age with suspected cryptosporidiosis (at least 2 samples/patient/3-day period) were obtained from local health department and public hospitals in South Bohemia during 2005–2007. Samples were examined for *Cryptosporidium* oocysts by using aniline-carbol-methyl violet staining and light microscopy at ×1,000 magnification (6). The microscopically positive samples were confirmed by DNA sequencing of the small subunit (SSU) rRNA gene. Total DNA was extracted from 200–300 mg stool by using the QIAamp DNA Stool Mini Kit (QIAGEN, Hilden, Germany), following the manufacturer’s instructions, after previous homogenization and disruption of oocysts with the Mini-BeadBeater (Biospec Products, Bartlesville, OK, USA). An ∼830-bp fragment of the SSU rRNA gene was amplified by nested PCR according to Jiang et al. (7). Purified PCR products were sequenced in both directions on an ABI3130 sequencer analyzer (Applied Biosystems, Foster City, CA, USA) by using the secondary PCR primers and the BigDye Terminator v3.1 Cycle Sequencing Kit (Applied Biosystems). Sequences were assembled by using Chromas Pro (www.techneleyum.com/-chromas.html) and aligned with reference sequences using ClustalX (ftp://ftp-igbmc.u-strasbg.fr/pub/ClustalX). The BLAST server (www.ncbi.nlm.nih.gov/BLAST) was used for DNA database searches. The SSU rRNA gene partial sequences of the 7 patient isolates have been submitted to GenBank (Table).

Of the 203 patients, 7 (3.4%) (6 children and 1 adult) had positive results for *Cryptosporidium* spp. Moreover, all samples obtained from these persons during the 3-day period were *Cryptosporidium* spp. positive; partial sequences of the *Cryptosporidium* SSU rRNA gene were obtained from all positive samples identifying 3 different species or genotypes of *Cryptosporidium*. Five were *C. parvum* (bovine genotype), I was *C. hominis*, and I contained the *Cryptosporidium* pig genotype II (Table). *Cryptosporidium* pig genotype II was found in stool samples from a 29-year-old man who also was infected with *Giardia intestinalis* (assemblage A) (data not shown).

Only *C. parvum* (bovine genotype), *C. hominis*, and *Cryptosporidium* rabbit genotype have been implicated in waterborne outbreaks of cryptosporidiosis in humans. Further studies are needed to determine the potential of other cryptosporidia of animal origin. Recent genetic and biologic characterization studies have identified 2 distinct host-adapted cryptosporidia in pigs, *C. suis* and *Cryptosporidium* pig genotype II. Furthermore, both above-mentioned cryptosporidia have been identified in untreated water (8). Pigs could be sources of *Cryptosporidium* water and food pollution and a consequent risk to public health.

Although human infection with *C. suis* has been previously described (9), human infection with *Cryptosporidium* pig genotype II has been never reported. This genotype was found in diarrheal stool of 1 adult patient in this study. However, onset of diarrhea could have been caused by co-infection with *G. intestinalis* (assemblage A), which recently also has been described in pigs (10). Contact with infected animals and ingestion of contaminated food or water could be