

Address for correspondence: Lan Anh Nguyen Thi, Laboratory for Molecular Diagnostics, National Institute of Hygiene and Epidemiology, Ha Noi, Vietnam; email: lananhnguyen@nihe.org.vn

Cholera in Yangon, Myanmar, 2012–2013

Wah Wah Aung, Kazuhisa Okada, Mathukorn Na-Ubol, Wirongrong Natakathung, Toe Sandar, Nan Aye Thidar Oo, Mya Mya Aye, Shigeyuki Hamada

Author affiliations: Ministry of Health Department of Medical Research (Lower Myanmar), Yangon, Myanmar (W.W. Aung, N.A.T Oo, M.M. Aye); Thailand-Japan Research Collaboration Center on Emerging and Re-emerging Infections, Nonthaburi, Thailand (K. Okada, M. Na-Ubol, W. Natakathung, S. Hamada); Osaka University Research Institute for Microbial Diseases, Osaka, Japan (K. Okada, S. Hamada); University of Medicine, Yangon (T. Sandar)

DOI: <http://dx.doi.org/10.3201/eid2103.141309>

To the Editor: *Vibrio cholerae* O1, a causative agent of cholera, is classified into 2 biotypes, classical and El Tor. Since 1817, cholera has spread from the Indian subcontinent to other regions of the globe 7 times (1). However, little information on the occurrence of cholera and *V. cholerae* in Myanmar has been published. Here, we report cholera cases and characterization of 58 clinical isolates of *V. cholerae* O1 serotype Ogawa recovered from patients with diarrhea in Yangon, Myanmar, during February 2012–June 2013.

During February and August 2012, rectal swab specimens were collected from patients suspected of having cholera in 4 hospitals in Yangon: New Yangon General Hospital, North Okkalapa General Hospital, Thingangyun Sanpya General Hospital, and Yangon Children Hospital. The specimens were cultured on thiosulfate citrate bile salts sucrose agar plates. After overnight incubation, several colonies that resembled to those of *V. cholerae* were confirmed as serogroup O1 by using slide agglutination tests with specific monoclonal antibodies (2).

Of the tested specimens, 34 isolates carried the *tcpA* gene, encoding the structural subunit of toxin-coregulated pilus, and the *rstR* gene, the repressor gene in the cholera toxin encoding (CTX) phage (3); these results may indicate that these strains belonged to the El Tor biotype. However, identification of the sequence type of the cholera toxin B subunit gene in these isolates revealed that they were of classical type (*ctxB^{Cl}*). Thus, these isolates were classified as atypical El Tor *V. cholerae* O1 (4)

carrying *ctxB^{Cl}* and *rstR^{El}*. Currently, the predominant clones causing cholera in Asia and Africa are atypical El Tor *V. cholerae*, CIRS101, and CIRS101-like variants (5,6). Myanmar isolates from 2012 and the CIRS101 strain contained a single nucleotide polymorphism in the *tcpA* gene at nt 266 (A→G) of the prototype seventh pandemic El Tor (N16961) strain.

Pulsed-field gel electrophoresis (PFGE) (7) using the 2012 isolates revealed 9 patterns (Table, <http://wwwnc.cdc.gov/EID/article/21/3/14-1309-T1.htm>). During the initial phase of cholera occurrences, *V. cholerae* O1 was mainly isolated from adults, and 9 pulsotypes were observed. During the later period (May–August), most isolates were from children <5 years of age, and pulsotype Y6 predominated.

We carried out multilocus variable-number tandem-repeat analysis (MLVA) (8) of the 2012 isolates to resolve distinct populations. MLVA yielded 13 isolate types, and all 18 isolates of pulsotype Y6 exhibited either MLVA profile 11.6.6.17.17 or a closely related profile that differed only by 1 repeat number. These data suggest that cholera was contracted mainly in adults and was caused by multiclonal *V. cholerae* O1. However, in children, *V. cholerae* has transformed from single clonal expansion since May 2012.

During March–June 2013, we extended our studies to characterize cholera organisms isolated from patients with severe diarrhea who were admitted to the original 4 hospitals as well as 2 additional hospitals, Yankin Children Hospital and Insein General Hospital. Of 24 cases, 16 patients showed symptoms of severe dehydration, including 1 patient who experienced shock. Other common symptoms in this patient population included fever (50%, 12/24), vomiting (92%, 22/24), and abdominal pain (33%, 8/24). Although fever is less common among patients with cholera-associated diarrhea (9), the frequency of fever was considerably high in this study. PFGE revealed 23 of the 24 isolates were identical to pulsotype Y7, which was the second-most prevalent pattern in 2012; MLVA profiles were also similar to those from 2012. Thus, the occurrences of cholera in 2013 may have been related to persistent transmission of a clone from 2012.

According to surveillance records from the Yangon Regional Health Center, the reported number of diarrhea cases in Yangon increased from 11,651 in 2010 and 11,016 in 2011 to 15,540 in 2012 and 13,919 in 2013. Although there were no reports of cholera outbreaks in Yangon, PFGE/MLVA results revealed that most of the cholera cases in this study were caused by isolates belonging to identical or closely related types. Thus, cholera outbreaks could have occurred in Yangon, and the related clone may have persisted.

In Myanmar, the illness rate for severe diarrhea is estimated to be 2.6–3.5 per 100,000 persons and the mortality

rate is 0.04–0.1 per 100,000 (10). In this study, the detection rates of *V. cholerae* O1 in stools from patients with severe diarrhea were 23% (49/213 cases) in 2012 and 14% (35/250 cases) in 2013, respectively. Although our investigation is merely the tip of the iceberg for studies of cholera in Myanmar, our data provide crucial initial insights into the genetic backgrounds of recent Yangon isolates of *V. cholerae* O1. Epidemiologic surveillance linked to laboratory investigations is need to minimize the risk for *V. cholerae* infection in children.

References

1. Sack DA, Sack RB, Nair GB, Siddique AK. Cholera. *Lancet*. 2004;363:223–33. [http://dx.doi.org/10.1016/S0140-6736\(03\)15328-7](http://dx.doi.org/10.1016/S0140-6736(03)15328-7)
2. Albert MJ, Ansaruzzaman M, Bardhan PK, Faruque ASG, Faruque SM, Islam MS, et al. Large epidemic of cholera-like disease in Bangladesh caused by *Vibrio cholerae* O139 synonym Bengal. *Lancet*. 1993;342:387–90. [http://dx.doi.org/10.1016/0140-6736\(93\)92811-7](http://dx.doi.org/10.1016/0140-6736(93)92811-7)
3. Bhattacharya T, Chatterjee S, Maiti D, Bhadra RK, Takeda Y, Nair GB, et al. Molecular analysis of the *rstR* and *orfU* genes of the CTX prophages integrated in the small chromosomes of environmental *Vibrio cholerae* non-O1, non-O139 strains. *Environ Microbiol*. 2006;8:526–634. <http://dx.doi.org/10.1111/j.1462-2920.2005.00932.x>
4. Safa A, Nair GB, Kong RY. Evolution of new variants of *Vibrio cholerae* O1. *Trends Microbiol*. 2010;18:46–54. <http://dx.doi.org/10.1016/j.tim.2009.10.003>
5. Grim CJ, Hasan NA, Taviani E, Haley B, Chun J, Brettin TS, et al. Genome sequence of hybrid *Vibrio cholerae* O1 MJ-1236, B-33, and CIRS101 and comparative genomics with *V. cholerae*. *J Bacteriol*. 2010;192:3524–33. <http://dx.doi.org/10.1128/JB.00040-10>
6. Reimer AR, Van Domselaar G, Stroika S, Walker M, Kent H, Tarr C, et al. Comparative genomics of *Vibrio cholerae* from Haiti, Asia, and Africa. *Emerg Infect Dis*. 2011;17:2113–21. <http://dx.doi.org/10.3201/eid1711.110794>
7. Okada K, Roobthaisong A, Nakagawa I, Hamada S, Chantaroj S. Genotypic and PFGE/MLVA analyses of *Vibrio cholerae* O1: geographical spread and temporal changes of isolates during the 2007–2010 cholera outbreaks in Thailand. *PLoS ONE*. 2012;7:e30863. <http://dx.doi.org/10.1371/journal.pone.0030863>
8. Stine OC, Alam M, Tang L, Nair GB, Siddique AK, Faruque SM, et al. Seasonal cholera from multiple small outbreaks, rural Bangladesh. *Emerg Infect Dis*. 2008;14:831–3. <http://dx.doi.org/10.3201/eid1405.071116>
9. Fukuda JM, Yi A, Chaparro L, Campos M, Chea E. Clinical characteristics and risk factors for *Vibrio cholerae* infection in children. *J Pediatr*. 1995;126:882–6. [http://dx.doi.org/10.1016/S0022-3476\(95\)70201-6](http://dx.doi.org/10.1016/S0022-3476(95)70201-6)
10. Than-Htain-Win. Effectiveness of oral cholera vaccination on prevention and control of severe diarrhoea disease in high risk area. In: *Proceedings of Symposium on Effects of Environmental Changes on Health*. Yangon (Myanmar): Myanmar Health Research Congress 2010; 2011. p. 40–50.

Address for correspondence: Kazuhisa Okada, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan; email: kazuhisa@biken.osaka-u.ac.jp

Role of Race/Ethnicity in Pulmonary Nontuberculous Mycobacterial Disease

Benjamin S. Thomas, Koh Okamoto

Author affiliations: Washington University School of Medicine, St. Louis, Missouri, USA (B.S. Thomas); Rush University Medical Center, Chicago, Illinois, USA (K. Okamoto)

DOI: <http://dx.doi.org/10.3201/eid2103.141369>

To the Editor: We read with interest the study of gender and age in nontuberculous mycobacterial (NTM) lung disease case-patients in Taiwan (1). NTM lung disease is relatively uncommon; however, the exact prevalence of NTM lung disease and causative organisms are largely unknown in many regions of the United States because the disease is not reportable. A recent study using Medicare claims data in the United States showed that the annual prevalence of NTM lung disease increased from 20 cases/100,000 persons in 1997 to 47 cases/100,000 persons in 2007 (2). The study also showed that Hawaii had the highest period prevalence of cases (396 cases/100,000 persons), which was at least partially attributed to the large Asian/Pacific Islander population (2). During June–December 2011, we conducted a cross-sectional study to evaluate the epidemiologic and clinical significance of NTM isolated from patients in Honolulu, Hawaii; the patients had suspected pulmonary tuberculosis (TB) and were in airborne isolation at a university-affiliated, tertiary-care hospital.

NTM cases were defined according to the 2007 criteria of the American Thoracic Society/Infectious Diseases Society of America (3). The process required to establish a diagnosis of NTM lung disease is sometimes lengthy; thus, patients who did not initially meet the disease criteria but who had cultures positive for NTM were reviewed again 1 year after the original data were collected to see if follow-up microbiological and radiographic studies would confirm the presence of NTM lung disease. Descriptive statistics were used to describe categorical and continuous variables. During June–December 2011, a total of 113 patients with suspected pulmonary TB were placed into isolation at the tertiary-care hospital. Of these patients, 85 (75.2%) were men and 28 (24.8%) were women; the median age was 59.8 ± 17 years. Eighteen (15.9%) patients were white, 92 (81.4%) were Asian/Pacific Islander, and 1 (0.9%) was African American; for 2 (1.8%) patients, race/ethnicity was classified as not specified/other.

Of the 113 isolated patients, 21 (18.6%) were positive for mycobacteria. Of these 21 patients, 14 (66.7%) were men and 7 (33.3%) were women; the median age