use antimicrobial drugs or proton pump inhibitors and had no known contacts with persons with diarrhea. He was mechanically ventilated and received oral vancomycin, intravenous metronidazole, and vasopressors. He died of respiratory failure and septic shock. In comparison to the patients described by Sandhu et al., the patient we report was younger and did not have a history of antimicrobial use.

SARS-CoV-2 has multifaceted presentations. Angiotensin-converting enzyme 2 receptor, which can act as a receptor for severe acute respiratory syndrome coronavirus, is expressed not only in alveolar cells but also in the gastrointestinal tract, including colonic cells (*2*,*3*). Diarrhea associated with COVID-19 might erode the normal microbial flora of the gut, leading to increased risk for CDI. Also, COVID-19 might weaken the immune system, leaving the patient vulnerable to CDI. COVID-19 patients produce inadequate interferon-γ and have defective macrophage activation and function, resulting in a dysregulated immune response (*4*). Interleukin-12 and interferon-γ are components of cell-mediated immunity. Interferon-γ produced by T-helper cells induces macrophages to destroy bacteria such as *C. difficile* (*5*).

The relationship between SARS-CoV-2 and CDI is still poorly understood. CDI might be a complication of COVID-19; however, we could not exclude the possibility of co-occurrence of CDI with COVID-19. Physicians should consider CDI when encountering a COVID-19 patient with diarrhea.

References

1. Sandhu A, Tillotson G, Polistico J, Salimnia H, Cranis M, Moshos J, et al. *Clostridiodes difficile* in COVID-19 patients, Detroit, Michigan, USA, March–April 2020. Emerg Infect Dis. 2020 May 22 [Epub ahead of print]. https://doi.org/10.3201/eid2609.202126
2. Li W, Moore MJ, Vasilieva N, Sui J, Wong SK, Berne MA, et al. Angiotensin-converting enzyme 2 is a functional receptor for the SARS coronavirus. Nature. 2003;426:450–4. https://doi.org/10.1038/nature02145
3. Hamming I, Timens W, Bulthuis MLC, Lely AT, Navis G, van Goor H. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis. J Pathol. 2004;203:631–7. https://doi.org/10.1002/path.1570
4. Blanco-Melo D, Nilsson-Payant BE, Liu WC, Uhl S, Hoagland D, Møller R, et al. Imbalanced host response to SARS-CoV-2 drives development of COVID-19. Cell. 2020; 181:1036–1045.e9. https://doi.org/10.1016/j.cell.2020.04.026
5. Cruz-Adalia A, Veiga E. Close encounters of lymphoid cells and bacteria. Front Immunol. 2016;7:405. https://doi.org/10.3389/fimmu.2016.00405

Address for correspondence: Kok Hoe Chan, Saint Michael's Medical Center, Newark, NJ 07101, USA; email: kchan2@primehealthcare.com

Zika Virus Infection, Philippines, 2012

Corazon Cerilla Buerano, Lady-Anne Suarez Pangilinan, Maria Terrese Alonzo Dimamay, Cynthia Abad Mapua, Mark Pierre Sijo Dimamay, Ronald Roll Matias, Filipinas Florendo Natividad, Maria Luisa de Guzman Daroy, Futoshi Hasebe, Kouichi Morita, Meng Ling Moi

Author affiliations: St. Luke's Medical Center, Quezon City, Philippines (C.C. Buerano, L.-A.S. Pangilinan, M.T.A. Dimamay, C.A. Mapua, M.P.S. Dimamay, R.R. Matias, F.F. Natividad); St. Luke's Medical Center College of Medicine, Quezon City (M.L.G. Daroy); WHO Collaborating Centre for Reference and Research on Tropical and Emerging Viral Diseases, Institute of Tropical Medicine (NEKKEN), Nagasaki, Japan (F. Hasebe, K. Morita, M.L. Moi)

DOI: https://doi.org/10.3201/eid2609.190896

To the Editor: Alera et al. described a 2012 case of Zika virus infection in the Philippines (*1*). In 2007, a Zika virus outbreak occurred in Yap, Micronesia, possibly caused by travelers from the Philippines (*2*). Zika virus infections were reported in the Philippines in 1953, 2012, and 2016 (*3*). Although frequent travel exchange between Yap and the Philippines could be a possible transmission route, no data on Zika virus infection were recorded in the Philippines between 1953 and 2012.

We detected Zika virus infection in 1 (0.75%) of 134 febrile, non–dengue infected patients at St. Luke's Medical Center (Quezon City, the Philippines) during 2010–2015 by subjecting patient serum samples to serological and molecular tests. Ethics clearance (reference no. 19042) for this study was given by St. Luke's Medical Center Institutional Ethics Review Committee. The only patient who tested positive for Zika virus was a 31-year-old woman diagnosed with an upper respiratory tract infection in 2010. Because of her work, she might not have traveled internationally. We obtained her serum sample on day 3 of fever. She did not have a rash or arthralgia. Although we did not isolate Zika virus according to guidelines (*4*), we confirmed infection using other techniques. The patient's serum sample tested positive for Zika virus RNA, IgM against Zika virus, and neutralizing antibodies against Zika virus by using a plaque reduction neutralization test to neutralize 50% of plaques ($PRNT_{50}$) ($PRNT_{50}$ Zika virus = 1:80, $PRNT_{50}$ dengue virus serotypes 1–4 <1:10). The sample tested negative for IgM against dengue and Japanese encephalitis viruses but

positive for IgG against Zika virus nonstructural protein 1. These results suggest local Zika virus infection in the Philippines since at least 2010, 2 years earlier than the previously reported infection (*1*).

This work was supported by the Agency for Research and Development (AMED) under grant no. AMED JP-20wm0125006, AMED Research on Emerging and Re-emerging Infectious Diseases (grant nos. 19fk0108109h0001, 20fk0108109h0001, 20fk0108123h1101) and the Joint Usage/Research Center on Tropical Disease, Institute of Tropical Medicine, Nagasaki University (grant no. 2020-Ippan-21). Partial support came from the Research and Biotechnology Group, St. Luke's Medical Center, Quezon City, Philippines (project no. 07-024).

References

1. Alera MT, Hermann L, Tac-An IA, Klungthong C, Rutvisuttinunt W, Manasatienkij W, et al. Zika virus infection, Philippines, 2012. Emerg Infect Dis. 2015;21:722–4. https://doi.org/10.3201/eid2104.141707
2. Musso D, Gubler DJ. Zika Virus. Clin Microbiol Rev. 2016;29:487–524. https://doi.org/10.1128/CMR.00072-15
3. Duong V, Dussart P, Buchy P. Zika virus in Asia. Int J Infect Dis. 2017;54:121–8. https://doi.org/10.1016/j.ijid.2016.11.420
4. Centers for Disease Control and Prevention. Testing for Zika virus infection. 2019 Jun 13 [cited 2020 Jun 11]. https://www.cdc.gov/zika/laboratories/types-of-tests.html

Address for correspondence: Meng Ling Moi, Department of Virology, Institute of Tropical Medicine, Nagasaki University, 1-12-4 Sakamoto Nagasaki, Nagasaki, Nagasaki 852-8523, Japan; email: sherry@nagasaki-u.ac.jp

CORRECTION

Correction: Vol. 26, No. 7

The name of author Xiankun Zeng was misspelled in in Changes in Approach to Cataract Surgery in an Ebola Virus Disease Survivor with Prior Ocular Viral Persistence (J.R. Wells et al.). The article has been corrected online (https://wwwnc.cdc.gov/eid/article/26/7/19-1559_article).