**To the Editor:** Ryu et al. reviewed international travel–related measures for pandemic influenza, including screening travelers for infection (*1*). Although the authors did not review the performance of individual screening tools, Ryu et al. reported that no evidence exists to indicate that screening has any substantial effect on preventing the spread of pandemic influenza.

However, government officials continue to call for international airport screening guidelines as a crucial measure to control coronavirus disease. Therefore, differentiating between screening tools with poor technical performance and those approved for fever detection is worthwhile. For example, the Food and Drug Administration (FDA) states that thermal scanners should not be used as standalone tools for fever detection (*2*). FDA instead recommends that officials use handheld infrared thermometers as screening tools.

Thermal scanners use long-wave infrared to generate heat map images of persons and objects. This technology records surface temperature; however, fever determination requires a measurement of core body temperature. A study with 1,109 participants showed a correlation with core temperature of merely $R^2 = 0.41$ for the most commonly used thermography region, the forehead (*3*). Performance of $R^2 = 0.69$ was achieved only with overlaid standard camera video and complex free-form deformation models. Participants were assessed individually, after being seated for 15 minutes, without topical cosmetics or eyewear, at a stable ambient temperature and humidity, and without nearby infrared radiation sources. These conditions are rarely, if ever, met in the airport setting.

Despite this evidence, costly thermal scanners have been deployed at airports in many countries. In contrast, inexpensive infrared thermometers are FDA approved for core temperature approximation. At their current performance, thermal scanners must be clearly distinguished from infrared thermometers, and thermal scanning should not be recommended for fever screening.

### References


1. Ryu S, Gao H, Wong JY, Shiu EYC, Xiao J, Fong MW, et al. Nonpharmaceutical measures for pandemic influenza in nonhealthcare settings—international travel-related measures. Emerg Infect Dis. 2020;26:961–6. https://doi.org/10.3201/eid2605.190993
2. US Food and Drug Administration. Enforcement policy for telethermographic systems during the coronavirus disease 2019 (COVID-19) public health emergency. 2020 Apr 16 [cited 2020 May 1] https://www.fda.gov/regulatory-information/search-fda-guidance-documents/enforcement-policy-telethermographic-systems-during-coronavirus-disease-2019-covid-19-public-health
3. Zhou Y, Ghassemi P, Pfefer J, Casamento J, McBride D, Chen M, et al. Large-scale clinical study of 'point of care' thermal imaging for febrile patient detection: towards optimal non-contact diagnostics in disease pandemics. In: Gannot I, editor. Proceedings volume 10872: optical fibers and sensors for medical diagnostics and treatment applications XIX. SPIE BiOS 10872; 2019 Mar 4; San Francisco, CA, USA. Bellingham (WA): Society of Photo-Optical Instrumentation Engineers; 2019 [cited 2020 May 1] https://www.spiedigitallibrary.org/conference-proceedings-of-spie/10872/108720X/Large-scale-clinical-study-of-point-of-care-thermal-imaging/10.1117/12.2514766.short?SSO=1


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# *Clostridioides difficile* in COVID-19 Patients, Detroit, Michigan, USA, March–April 2020

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**To the Editor:** Sandhu et al. (*1*) reported 9 patients who were co-infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and *Clostridioides difficile*. *C. difficile* infection (CDI) can be a co-occurrence or result of antimicrobial drug overuse and is potentially a complication of coronavirus disease (COVID-19). We report a 52-year-old man with hypertension who had fever, respiratory symptoms, abdominal pain, and diarrhea for 3 days. At admission to Saint Michael's Medical Center (Newark, New Jersey, USA), he had a temperature of 101.8°F but was otherwise hemodynamically stable. He had an elevated absolute lymphocyte count (700 cells/μL), indicating lymphopenia. He tested positive for SARS-CoV-2 RNA by reverse transcription PCR and had elevated inflammatory markers on blood profile. He tested positive for *C. difficile* toxin and antigen at admission. He did not

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


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# Zika Virus Infection, Philippines, 2012

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**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