cured without valve surgery; it was cured with a 30-month antimicrobial drug regimen (10).

The role for serial serologic testing in assessing cure of Bartonella endocarditis is unknown. In our cases, as in a previous report (10), a drop in Bartonella titers occurred over a 3-year period in those who were cured, suggesting follow-up serologic testing might be useful to assess Bartonella endocarditis clinical cure.

Our findings suggest that a simple, inexpensive drug regimen is optimal therapy for Bartonella endocarditis and that serial serologic testing can confirm adequate treatment and cure. Further research is needed to validate this approach to managing Bartonella endocarditis.

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References

1. Spach DH, Callis KP, Pauw DS, Houze YB, Schoenknecht FD, Welch DF, et al. Endocarditis caused by Rochalimaea quintana in a patient infected with human immunodeficiency virus. J Clin Microbiol. 1993;31:692–4.

2. Tattevin P, Watt G, Revest M, Arvieux C, Fournier PE. Update on blood culture-negative endocarditis. Med Mal Infect. 2015;45:1–8. http://dx.doi.org/10.1016/j.medmal.2014.11.003

3. Fournier P-E, Thuny F, Richet H, Lepidi H, Casalta JP, Arzouni JP, et al. Comprehensive diagnostic strategy for blood culture-negative endocarditis: a prospective study of 819 new cases. Clin Infect Dis. 2010;51:131–40. http://dx.doi.org/10.1086/653675

4. Chalonier GL, Harrison TG, Birles RJ. Bartonella species as a cause of infective endocarditis in the UK. Epidemiol Infect. 2013;141:841–6. http://dx.doi.org/10.1017/S0950268812001185

5. Edouard S, Nabet C, Lepidi H, Fournier PE, Raoult D. Bartonella, a common cause of endocarditis: a report on 106 cases and review. J Clin Microbiol. 2015;53:824–9. http://dx.doi.org/10.1128/JCM.02827-14

6. Klein JL, Nair SK, Harrison TG, Hunt I, Fry NK, Friedland JS. Prosthetic valve endocarditis caused by Bartonella quintana. Emerg Infect Dis. 2002;8:202–3. http://dx.doi.org/10.3201/eid0802.010206

7. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zotti F, et al. 2015 ESC guidelines for the management of infective endocarditis. Eur Heart J. 2015;36:3075–128. http://dx.doi.org/10.1093/eurheartj/ehv319

8. Raoult D, Fournier PE, Vandenesch F, Mainardi JL, Eykyn SJ, Nash J, et al. Outcome and treatment of Bartonella endocarditis. Arch Intern Med. 2003;163:226–30. http://dx.doi.org/10.1001/archinte.163.2.226

9. Rolaín JM, Brouqui P, Koehler JE, Maguina C, Dolan MJ, Raoult D. Recommendations for treatment of human infections caused by Bartonella species. Antimicrob Agents Chemother. 2004;48:1921–33. http://dx.doi.org/10.1128/AAC.48.6.1921-1933.2004

10. Lespruit P, Noël V, Chazourillères P, Brun-Buisson C, Deforges L. Cure of Bartonella endocarditis of a prosthetic aortic valve without surgery: value of serologic follow-up. Clin Microbiol Infect. 2003;9:239–41. http://dx.doi.org/10.1046/j.1469-0691.2003.00509.x

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Zika Virus Infection and Prolonged Viremia in Whole-Blood Specimens

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We tested whole-blood and plasma samples from immunocompetent patients who had had benign Zika virus infections and found that Zika virus RNA persisted in whole blood substantially longer than in plasma. This finding may have implications for diagnosis of acute symptomatic and asymptomatic infections and for testing of blood donations.

Since cases of severe neurologic disorders among adults (1) and fetal abnormalities (2) linked to Zika virus infections were initially reported, the World Health Organization has deemed the Zika virus outbreak a “public health emergency of international concern” and has raised Zika virus to the same level of concern as Ebola virus. In response, medical authorities from many countries have released advice and guidelines regarding prevention and diagnosis to contain the spread of this virus and guidelines regarding safety of whole blood and blood components. In August 2016, the Food and Drug Administration announced universal testing for Zika virus RNA in donated whole blood and blood components taken in the United States and its territories using a qualitative molecular assay on plasma specimens (3).

In Europe, advice on Zika virus regarding the safety of substances of human origin (4) has been applied in France since February 15, 2016. A qualitative individual molecular test for Zika virus RNA in plasma specimens is being used on whole-blood specimens from blood donors living in Guadeloupe and Martinique, 2 overseas administrative areas where Zika virus is autochthonous. Furthermore, in mainland France and in French overseas areas where no active Zika virus transmission exists, and since the beginning of the Zika virus outbreak in 2015, blood donors who have recently visited areas or countries with ongoing Zika
virus transmission are subject to a 28-day temporary deferral after their departure from these areas, a period twice the assumed maximum incubation period for Zika virus. Similarly, temporary deferral applies to blood donors who have a sex partner who has been recently infected or potentially exposed to a confirmed or suspected Zika virus infection within the previous 3 months.

We report results from a 2016 longitudinal follow-up of Zika virus RNA quantification in EDTA whole-blood and plasma samples taken from 5 immunocompetent patients (2 men, 33 and 70 years of age, and 3 women, 55, 58, and 67 years of age) and results from a point-to-point comparison of Zika viral loads on both EDTA whole-blood and corresponding plasma samples (27 pairs). We extracted RNA by using the MagNA Pure 96 instrument with the DNA and Viral NA Small Volume Kit (Roche Diagnostics, Meylan, France) (input and output volumes 200 and 100 µL). We quantified RNA by using the RealStar Zika RNA RT-PCR kit 1.0 (Altona Diagnostics GmbH, Hamburg, Germany) (limit of selection 2.48 log copies/mL). We always successfully detected the manufacturer’s internal control. All samples were collected from patients who had returned from the Caribbean or South and Central America and had had a benign form of Zika virus infection.

Results from the follow-up (18 whole-blood and 21 plasma samples) showed that the median duration of Zika virus was 22 (range 14–100) days in whole blood and 10 (range 7–37) days in plasma (p = 0.058). Mean viral loads of positive samples were 3.39 log copies/mL in whole blood (n = 13) and 2.52 log copies/mL in plasma (n = 6; p = 0.001). Viral loads in the last positive samples varied from 2.7 to 3.9 log copies/mL in whole blood and 2.2 to 2.8 log copies/mL in plasma (p = 0.06). Whole-blood samples from 2 patients remained positive at 14 and 63 days after their plasma samples had become negative (Figure, panel A).

The point-to-point comparison (18 pairs from the follow-up and 9 additional pairs) showed that Zika virus RNA was quantifiable in 23 whole-blood specimens but in only 10 plasma samples. Mean viral load was 3.50 (range 2.75–4.17) log copies/mL in whole blood and 3.01 (range 2.21–4.10) log copies/mL in plasma (p = 0.018) (Figure, panel B).

These data show that Zika virus RNA persisted in whole blood after it disappeared in plasma. Similar results have been reported previously for West Nile virus, also a member of the *Flaviviridae* family (5,6), and for Zika virus with a qualitative in-house PCR (7).

Our data have 3 main consequences. First, for acute symptomatic infection, the use of whole blood extends the period of diagnosis. Second, for asymptomatic infections with a high likelihood of low viral load, virus detection in plasma might be less sensitive than detection in whole-blood specimens. Third, according to our data that show that viremia can persist for >28 days after symptom onset, recommendations used to reduce the risk for Zika virus transmission through blood or blood components should be modified. Potential options such as extending the deferral period or testing blood donations for Zika virus RNA in whole blood should be considered.
Overall, our data show that use of whole-blood specimens is much more sensitive than use of plasma samples to detect Zika virus RNA. These data could be useful in recommending the use of whole blood instead of plasma for the molecular diagnosis of acute symptomatic and asymptomatic Zika virus infections and for the safety of whole blood and blood components from donors, as well as for the safety of organs, tissues, and cells from deceased and living donors.

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References
1. Oehler E, Watrin L, Larre P, Leparc-Goffart I, Lastere S, Valour F, et al. Zika virus infection complicated by Guillain-Barre syndrome—case report, French Polynesia, December 2013. Euro Surveill. 2014;19:20720. http://dx.doi.org/10.2807/1560-7917.ES2014.19.9.20720
2. Schuler-Faccini L, Ribeiro EM, Feitosa IM, Horovitz DD, Cavalcanti DP, Pessoa A, et al.; Brazilian Medical Genetics Society–Zika Embryopathy Task Force. Possible association between Zika virus infection and microcephaly—Brazil, 2015. MMWR Morb Mortal Wkly Rep. 2016;65:59–62. http://dx.doi.org/10.15585/mmwr.mm6503e2
3. US Department of Health and Human Services Food and Drug Administration Center for Biologics Evaluation and Research. Revised recommendations for reducing the risk of Zika virus transmission by blood and blood components—guidance for industry [cited 2016 Jul 30]. https://www.fda.gov/downloads/BiologicsBloodVaccines/GuidanceComplianceRegulatoryInformation/ScientificGuidances/Blood/UCM518213.pdf
4. European Center for Disease Prevention and Control. ECDC scientific advice: Zika virus and safety of human organs, tissues, and cells. A guide for preparedness activities in Europe [cited 2016 Aug 30]. http://ecdc.europa.eu/en/publications/_layouts/forms/Publication_DispForm.aspx?List=4f55ad51-4aed-4d32-b960-af70113db90&ID=1527
5. Lanteri MC, Lee TH, Wen L, Kaidarova Z, Bravo MD, Kiely NE, et al. West Nile virus nucleic acid persistence in whole blood months after clearance in plasma: implication for transfusion and transplantation safety. Transfusion. 2014;54:3232–41. http://dx.doi.org/10.1111/trf.12764
6. Rios M, Daniel S, Chancey C, Hewlett IK, Stramer SL. West Nile virus adheres to human red blood cells in whole blood. Clin Infect Dis. 2007;45:181–6. http://dx.doi.org/10.1086/518850
7. Lustig Y, Mendelson E, Paran N, Melamed S, Schwartz E. Detection of Zika virus RNA in whole blood of imported Zika virus disease cases up to 2 months after symptom onset, Israel, December 2015 to April 2016. Euro Surveill. 2016;21:30269. http://dx.doi.org/10.2807/1560-7917.ES.2016.21.26.30269

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Severe MRSA Enterocolitis Caused by a Strain Harboring Enterotoxins D, G, and I

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We describe a case of methicillin-resistant *Staphylococcus aureus* (MRSA) enterocolitis in a healthy adult with previous antibiotic exposure. Colonoscopy revealed diffuse colitis and mild ileitis without ulceration. Stool cultures demonstrated abundant growth of MRSA and absent normal flora. Oral vancomycin treatment was effective and seems to be the consensus choice for therapy.

*Staphylococcus aureus* was recognized as a cause of antibiotic-associated colitis (AAC) in the mid-20th century (1,2). *Clostridium difficile* was later identified as the primary cause of AAC, and appreciation of *S. aureus* as a potential etiology declined (2). Methicillin-resistant *S. aureus* (MRSA) has also been implicated as a cause of AAC, with most reports coming from Japan. We report a case of MRSA enterocolitis in Canada caused by a strain harboring multiple enterotoxins.

In 2014, a 22-year-old woman sought care after 10 days of acute and profuse diarrhea, abdominal cramping, nausea, and weight loss of 5 lbs. She had 10–30 bowel movements a day and had observed blood-tainted stool. The patient reported a history of migraine and depression but was otherwise healthy. She worked in a pet store and had not been hospitalized. In the previous 2 months, she had been treated for chlamydia with a single course of azithromycin and cefixime. Subsequently, she received oral ciprofloxacin to be started after stool collection. On March 6, she was admitted to the hospital with complaints of abdominal pain, nausea, and vomiting. She was afebrile; blood pressure was 104/58 mm Hg and pulse 91 bpm. Her abdomen was soft with normal bowel sounds except for mild distention. Tenderness was appreciated in the left lower quadrant without guarding or rebound. She was admitted to the hospital with a diagnosis of diarrhea.

Overnight, she developed severe abdominal pain with nausea and vomiting. She was hemodynamically unstable with blood pressure at 70/30 mm Hg and pulse 130 bpm. Overall, her abdomen was markedly distended with abdominal rigidity and rebound tenderness. Investigations revealed significant leukocytosis with a white blood cell count of 60,000 cells/μL, with 85% neutrophils. Abdominal computed tomography showed generalized peritoneal fat stranding, thickening of the anterior layer of the superior mesenteric vein, and significant ascites. She was transferred to the surgical intensive care unit with a diagnosis of Clostridium difficile–induced toxic megacolon.

The patient received intravenous fluids and ceftriaxone. She was noted to have a history of multiple gastrointestinal surgeries for adhesive disease and possible Crohn’s disease. She underwent a contrast-enhanced computed tomography scan of the abdomen and pelvis, which showed multiple small bowel and colonic perforations. She was taken to the operating room for an exploratory laparotomy with a subtotal colectomy. A distal ileostomy was created, and the small bowel was resected. Her postoperative course was complicated by a peritonitis, which required a subsequent laparotomy. She was discharged to an extended care facility after 3 months of hospitalization.

In 2005, a 16-year-old girl from Illinois presented with dysentery, fever, and abdominal pain. She was treated with oral vancomycin. On follow-up, she had symptoms of diarrhea and abdominal pain. She was treated with metronidazole and oral vancomycin. After treatment, she had fever, abdominal pain, and hypotension. She was admitted to the hospital and underwent surgery. The patient had a history of abdominal pain and diarrhea. She was noted to have a history of multiple gastrointestinal surgeries for adhesive disease and possible Crohn’s disease. She underwent a contrast-enhanced computed tomography scan of the abdomen and pelvis, which showed multiple small bowel and colonic perforations. She was taken to the operating room for a subtotal colectomy. A distal ileostomy was created, and the small bowel was resected. Her postoperative course was complicated by a peritonitis, which required a subsequent laparotomy. She was discharged to an extended care facility after 3 months of hospitalization.