amplified the variable 3' end of the B646L (p72) gene by using 2 oligonucleotide primers, p72-U and p72-D (*3*). To amplify the entire E183L gene (p54), we used primers described by Oviedo et al. (*8*), and to amplify the tetramer amino acid repeats within the hypervariable central variable region of the B602L gene, we used primers described by Gallardo et al. (*5*). Our results showed that sequences for all ASFV isolates from this outbreak and those for isolates previously collected in eastern Europe and eastern Africa were 100% homologous over the p72, p54, and central variable region gene-coding regions. The p72 sequences clustered in genotype II (Figure).

During the 2015 ASFV outbreak in Zimbabwe, a total of 3,427 pigs were at risk for infection in the affected area. Of those, 2,836 (\approx 83%) became infected, and all infected pigs died. The 591 pigs that did not become infected had been confined in pens and did not have exposure to infected pigs or their products. A follow-up study is under way in the region to genetically characterize the viruses in this outbreak, focusing on the p54, p30 and, central variable region genes.

All villages affected during the 2015 outbreak in Zimbabwe were along the northern border with Mozambique, where genotype II has been found before. It is essential that more of the ASFVs circulating in eastern and southern Africa be sequenced so that their relatedness can be determined. This knowledge will enable the establishment of an epidemiologic link between outbreaks in the region and underscore the need for adequate quarantine measures to prevent ASF from becoming endemic in southern and eastern Africa.

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References

- Penrith ML, Lopes Pereira C, Lopes da Silva MMR, Quembo C, Nhamusso A, Banze J. African swine fever in Mozambique: review, risk factors and considerations for control. Onderstepoort J Vet Res. 2007;74:149–60.
- Misinzo G, Magambo J, Masambu J, Yongolo MG, Van Doorsselaere J, Nauwynck HJ. Genetic characterization of African swine fever viruses from a 2008 outbreak in Tanzania. Transbound Emerg Dis. 2011;58:86–92. http://dx.doi.org/10.1111/ j.1865-1682.2010.01177.x
- Bastos ADS, Penrith M-L, Crucière C, Edrich JL, Hutchings G, Roger F, et al. Genotyping field strains of African swine fever virus by partial p72 gene characterisation. Arch Virol. 2003;148:693– 706. http://dx.doi.org/10.1007/s00705-002-0946-8
- Rowlands RJ, Michaud V, Heath L, Hutchings G, Oura C, Vosloo W, et al. African swine fever virus isolate, Georgia, 2007. Emerg Infect Dis. 2008;14:1870–4. http://dx.doi.org/10.3201/ eid1412.080591
- Gallardo C, Fernández-Pinero J, Pelayo V, Gazaev I, Markowska-Daniel I, Pridotkas G, et al. Genetic variation among African swine fever genotype II viruses, eastern and central Europe. Emerg Infect Dis. 2014;20:1544–7. http://dx.doi.org/10.3201/eid2009.140554

- Misinzo G, Kasanga CJ, Mpelumbe-Ngeleja C, Masambu J, Kitambi A, Van Doorsselaere J. African swine fever virus, Tanzania, 2010–2012. Emerg Infect Dis. 2012;18:2081–3. http://dx.doi.org/10.3201/eid1812.121083
- Lubisi BA, Dwarka RM, Meenowa D, Jaumally R. An investigation into the first outbreak of African swine fever in the Republic of Mauritius. Transbound Emerg Dis. 2009;56:178–88. http://dx.doi.org/10.1111/j.1865-1682.2009.01078.x
- Oviedo JM, Rodríguez F, Gómez-Puertas P, Brun A, Gómez N, Alonso C, et al. High level expression of the major antigenic African swine fever virus proteins p54 and p30 in baculovirus and their potential use as diagnostic reagents. J Virol Methods. 1997;64:27–35. http://dx.doi.org/10.1016/S0166-0934(96)02140-4

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Management of *Bartonella* Prosthetic Valve Endocarditis without Cardiac Surgery

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Two cases of *Bartonella* prosthetic valve endocarditis were cured when treated for 2 weeks with gentamicin and 3 months with doxycycline. Clinical cure correlated with decreased *Bartonella* antibody titers. This report suggests a strategy to monitor, treat, and cure *Bartonella* prosthetic valve endocarditis.

B actionella, a causative agent of blood culture-negative endocarditis (BCNE) that usually requires valve surgery, was first attributed to endocarditis over 20 years ago (1-4). We report 2 cases of *Bartonella* prosthetic valve endocarditis that were successfully treated with antimicrobial drugs alone.

RESEARCH LETTERS

Case 1 involved a 62-year-old man admitted to the hospital in 2011 with left-flank pain and a 3-year history of anorexia, weight loss, night sweats, and recent diagnosis of anemia. He kept pet cats. He underwent mechanical aortic valve replacement and patent foramen ovale closure in 1992 and a repeat aortic valve replacement and an aortic root replacement for a chronic type A dissection in 1996.

On physical examination, he was afebrile and had subconjunctival hemorrhages, normal prosthetic heart sounds, and 10-cm splenomegaly. Blood tests showed a hemoglobin concentration of 9 g/dL (reference range 14.0-17.5 g/dL), serum creatinine of 2.76 mg/dL (reference range 0.6-1.2 mg/dL), C-reactive protein of 48 mg/L (reference range 0.08-3.1 mg/L), rheumatoid factor of 742 U/mL (reference range 0-30 U/mL), and c-ANCA (cytoplasmic antineutrophil cytoplasmic antibody) positivity (proteinase 3 autoantibody concentration, 18.3 U/mL). Urine dipstick (Combur 7 Test; Roche Diagnostics Ltd, Basel, Switzerland) revealed the presence of blood (4+), and renal biopsy demonstrated necrotizing crescentic glomerulonephritis. Five blood cultures were sterile. Transesophageal echocardiography showed no evidence of endocarditis. Bartonella serologic testing was conducted with an indirect immunofluorescence assay by using the manufacturer's instructions (MRL, Cypress, CA, USA); results showed high IgG titers to both B. henselae and B. quintana (Table), but the infecting species could not be determined. The patient was treated for 2 weeks with intravenous gentamicin (2 mg/kg/d) and for 3 months with oral doxycycline (100 mg $2\times/d$). Nine months after completing treatment, he was well: splenomegaly had resolved, and hemoglobin (15.6 g/dL) and creatinine (1.31 mg/dL) concentrations approached normal levels. He was well when last reviewed in 2014.

Case 2 involved a 29-year-old woman with inflammatory bowel disease and primary sclerosing cholangitis. Her symptoms began in 2011 with fever, rigors, night sweats, and anorexia for 2 weeks. She had no pets but recalled contact with a kitten 8 months previously. In 2002, BCNE developed, requiring mechanical aortic and mitral valve replacements. In 2003, BCNE was again diagnosed but was complicated by an ascending aorta to left atrial fistula, requiring an aortic root replacement, a homograft, and a repeat mechanical mitral valve replacement.

Physical examination revealed fever, a splinter hemorrhage, and an ejection systolic murmur. Blood tests revealed a low hemoglobin concentration (10.9 g/dL), a high C-reactive protein concentration (26 mg/L), normal renal function, and positivity for rheumatoid factor (114 U/mL). Transesophageal echocardiograms revealed no evidence of endocarditis. One of 20 blood culture tests grew B. henselae after a 19-day incubation (Public Health England, identified by partial sequencing of 16S rDNA). A serologic test for Bartonella was strongly positive (Table). Three days after starting treatment with oral doxycycline (100 mg $2\times/d$) and intravenous gentamicin (3 mg/kg/d), she became afebrile; she received 14 days of gentamicin and 3 months of doxycycline in total. Two months later, she remained well, and her C-reactive protein concentration was <5 mg/L. Because of symptomatic stenosis caused by structural deterioration of the replacement aortic valve, she underwent another aortic valve and root replacement 19 months after completing antimicrobial drugs. No evidence of active endocarditis was found during surgery.

Bartonella antibody titers dropped slowly over a period of 3 years in both patients (Table). Only case 1 had definite infective endocarditis when using the modified Duke diagnostic criteria. However, because the Duke criteria are insensitive for BCNE diagnosis, it has been proposed that a *Bartonella* IgG titer of \geq 1:800 and a positive Western blot or PCR analysis when using valve or blood specimens should be considered major Duke criteria (5).

Most reported cases of *Bartonella* endocarditis involve native valves; the first prosthetic valve infection was reported in 2002 (6). Although >80% of patients require valve replacement, infection with *Bartonella* is not in itself a recognized indication for surgery. Because our patients responded to medication, we did not need to consider cardiac surgery. The optimal antimicrobial drug therapy and duration for *Bartonella* endocarditis is undetermined. The recommended regimen of gentamicin for 14 days and doxycycline for 4 weeks (7) has limited evidence supporting its use (8,9). We found only 1 case of *Bartonella* prosthetic valve endocarditis

Table. Bartonella antibody titers in cases 1 and 2 by month after diagnosis*				
Month after diagnosis	<i>B. henselae</i> IgM	<i>B. henselae</i> IgG	<i>B. quintana</i> IgM	<i>B. quintana</i> IgG
Case 1				
0	40	32,768	<20	1,024
8	<20	16,385	<20	512
11	20	8,096	<20	512
16	<20	8,096	<20	256
25	<20	4,096	<20	64
43	<20	2,048	<20	64
Case 2				
0	<20	8,192	<20	512
16	<20	≥512	<20	64
41	<20	64	<20	<64

*All time points were assayed in parallel. Antibody titers are the inverse of the greatest dilution that exhibited a reaction.

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

- Spach DH, Callis KP, Paauw 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.
- 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
- 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
- Chaloner GL, Harrison TG, Birtles 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
- 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
- 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
- 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
- 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
- Rolain 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
- Lesprit P, Noël V, Chazouillères 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.

 $S_{(1)}$ 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