The patient was successfully extubated. He was transferred to a pneumology ward on day 9 and discharged on day 15. Antimicrobial treatment was stopped on day 10.

Most nonhuman strains of *V. metschnikovii* are usually found in aquatic habitats (e.g., lakes and marine waters). Human clinical infections with this bacterium are rare; however, cases of epidemic diarrhea caused by *V. metschnikovii* have been reported (5,6). Contamination by water or fish was not always demonstrated in these cases, but an orofecal source is possible. In coproculture, this microorganism is probably not diagnosed: it was initially identified as normal aerobic flora because it was oxidase negative.

The first case of septicemia with *V. metschnikovii* was reported in 1981 in a patient with peritonitis and an inflamed gallbladder (1). Three other patients with similar septicemia, all >70 years of age, were described (7,8); 2 had polymicrobial results in blood cultures. *V. metschnikovii* was also found in a mucocutaneous site (wound infection) after saphenectomy in swab samples of the wound site (9).

The patient in our study denied contact with lake or sea water, and he had not eaten any seafood. He was a retired carpenter without contact with domestic or wild animals and did not recall an episode of diarrhea before his hospitalization. The source of contamination that caused his acute respiratory failure was not identified.

Miyake et al. showed that *V. metschnikovii* produces a cytolysin with hemolytic properties (10). This finding might explain the invasive process of this bacterium, which resulted in pulmonary lesions in a patient with respiratory deficiency. As far as we know, this is the first case of pneumonia caused by *V. metschnikovii*.

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References

1. Jean-Jacques W, Rajashekaraiah KR, Farmer JJ, Hickman FW, Morris JG, Kallick CA. *Vibrio metschnikovii* bacteremia in a patient with cholecystitis. J Clin Microbiol. 1981;14:711–2.
2. Lee JV, Donovan TJ, Furniss AL. Characterization, taxonomy, and emended description of *Vibrio metschnikovii.* Int J Syst Bacteriol. 1978;28:99–111.
3. Hansen W, Pohl P, Seynave D, Bughin J, Yourassowski E. Isolements de *Vibrio metschnikovii* en Belgique. Ann Med Vet. 1989;133:343–6.
4. Farmer JJ III, Hickman-Brenner FW, Fanning GR, Gordon CM, Brenner DJ. Characterization of *Vibrio metschnikovii* and *Vibrio gazogenes* by DNA-DNA hybridization and phenotype. J Clin Microbiol. 1988;26:1993–2000.
5. Dalsgaard A, Alarcon A, Lanata CF, Jensen T, Hansen HJ, Delgado F, et al. Clinical manifestations and molecular epidemiology of five cases of diarrhoea in children associated with *Vibrio metschnikovii* in Arequipa, Peru. J Med Microbiol. 1996;45:494–500.
6. Magalhaes V, Branco A, de Andrade Lima R, Magalhaes M. *Vibrio metschnikovii* among diarrheal patients during cholera epidemic in Recife, Brazil. Rev Inst Med Trop Sao Paulo. 1996;38:1–3.
7. Hansen W, Freney J, Benyagoub H, Letouzey MN, Gigi J, Wauters G. Severe human infections caused by *Vibrio metschnikovii.* J Clin Microbiol. 1993;31:2529–30.
8. Hardardottir H, Vikenes K, Digranes A, Lassen J, Halstensen A. Mixed bacteremia with *Vibrio metschnikovii* in a 83-year-old female patient. Scand J Infect Dis. 1994;26:493–4.
9. Linde HJ, Kobuch R, Jayasinghe S, Reischl U, Lehn N, Kaulfuss S, et al. *Vibrio metschnikovii*, a rare cause of wound infection. J Clin Microbiol. 2004;42:4909–11.
10. Miyake M, Honda T, Miwatani T. Purification and characterization of *Vibrio metschnikovii* cytolysin. Infect Immun. 1988;56:954–60.

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Lassa Fever, Nigeria, 2003 and 2004

To the Editor: Suspected outbreaks of Lassa fever have been reported in the northern part of Edo, Nigeria, including Ekpoma, Igarra, and Ibilo, in 2001 and between November 2003 and March 2004 (1,2). To confirm Lassa fever activity in this area, serum samples were collected at the Specialist Teaching Hospital in Irrua (ISTH) from September 2003 to January 2004. Approximately 16,000 patients are seen each year at ISTH, and ≈80% of them have febrile illness. Serum specimens were taken from patients with febrile illness (n = 31), healthy contact persons (n = 17), and healthy hospital staff (n = 12). The samples were analyzed by Lassa virus–specific reverse-transcriptase polymerase chain reaction (RT-PCR) at the University of Lagos. Aliquots of specimens were sent to the Bernhard-Nocht Institute (BNI in Hamburg, Germany) for confirmatory PCR analysis, serologic testing, and virus isolation. The PCR used at both facilities was based on primers 80F2 and 36E2 that targeted the glycoprotein precursor (GPC) gene (3), although the protocols were slightly different. At BNI, virus RNA was purified by QIAamp viral RNA kit (Qiagen, Hilden, Germany), and RT-PCR was performed with Superscript II RT/Platinum Taq polymerase 1-step reagents (Invitrogen, Karlsruhe, Germany). This PCR assay has a 95% detection limit of 2,500 copies/mL (4). At the University of Lagos, virus RNA purification and RT-PCR were performed with diatomaceous silica and Brilliant single-step RT-PCR kit (Stratagene, Heidelberg, Germany), respectively. Serologic testing for Lassa virus–specific immunoglobulin G (IgG) and IgM was performed by indirect immunofluorescence assay

(IFA) by using Vero cells infected with Lassa virus strain Josiah. Virus was isolated in the biosafety level 4 laboratory at BNI with Vero cells. Results of the tests are summarized in the Table.

Acute Lassa virus infection, as shown by a positive PCR result, was diagnosed at the University of Lagos in 1 patient. This result was independently confirmed at BNI, and 2 additional samples tested positive by PCR. The PCR signals were weak, which suggests that discrepancies between laboratories stem from higher sensitivity of the assay used at BNI. Presence of a low IgM titer in the absence of IgG in 2 of the PCR-positive samples is also consistent with an acute infection. Two of the Lassa virus–positive persons (04-02 and 04-10) had febrile illness that indicated symptomatic Lassa fever, while 1 (04-04) had been classified as an asympto-

matic contact at the time of sampling. Retrospective investigation showed no evidence of illness in this person before or after sampling. Sequencing the diagnostic PCR fragments (300 nucleotides [nt] of GPC gene) from the 3 patients indicated infections by closely related strains. The sequence of patient 04-10 (GenBank accession no. DQ010031) differed by 4% from those of patients 04-02 and 04-04, while the latter sequences were identical (GenBank accession no. DQ010030). The facts that patients 04-02 and 04-04 were sisters who lived in the same house, that their samples were taken on the same day (January 28, 2004), and that the sequences were identical suggest a common source of infection or an infection chain. The detection of an asymptomatic or mild Lassa virus infection in the contact person agrees with population-based studies in

Sierra Leone that show only 9%–26% of all Lassa virus infections are associated with fever (5).

In an additional 10 samples, IgM with or without IgG was detected, primarily in patients with febrile illness. IgG in the absence of IgM was detected in 1 contact and 4 healthcare workers. All serologic IFA findings were confirmed with μ-capture and IgG enzyme-linked immunosorbent assays developed at BNI. Virus isolation was attempted with all samples that tested positive by PCR or IgM IFA. Lassa virus was isolated from 1 PCR-positive serum (04-10). The strain was designated Nig04-010. To characterize Lassa virus circulating in north Edo, phylogenetic analysis was performed. In addition to the GPC sequences of the diagnostic PCR fragments, part of the L gene of Nig04-010 was amplified and sequenced (780 nt, GenBank accession no. AY693637). Phylogenetic analysis of these sequences showed that the virus circulating around Irrua belongs to phylogenetic lineage II, which comprises Lassa virus strains from the southeastern part of Nigeria (6). Thus, genotype and geographic origin of the viruses characterized here correspond.

These data provide evidence for Lassa fever activity in north Edo. Approximately 6% of febrile patients tested had PCR-confirmed Lassa fever, which extrapolates to hundreds of patients with Lassa fever per year, when one considers the number of patients with febrile illness seen at ISTH. As shown here and elsewhere, PCR is a useful tool to diagnose Lassa virus infection (3,7), a prerequisite for effective ribavirin treatment (8). First steps have been made to establish molecular diagnostics for Lassa virus at the University of Lagos. Further efforts are necessary to improve the laboratory infrastructure in the country.

Table. Lassa virus–specific findings in 60 serum samples from Irrua Specialist Teaching Hospital, Edo, Nigeria*

Patient	RT-PCR†	IgM titer‡	IgG titer‡
Patients with fever (n = 31)			
04-10	Positive§	–	–
04-02	Positive	1:40	–
04-51	–	1:160	–
04-34	–	1:40	–
04-03	–	1:>20,480	1:20,480
03-05	–	1:320	1:20,480
03-01	–	1:160	1:10,240
04-08	–	1:80	1:20,480
04-33	–	1:20	1:640
04-52	–	1:160	1:40
04-53	–	1:40	1:40
Contact persons (n = 17)			
04-04	Positive	1:20	–
03-04	–	1:160	1:>20,480
04-11	–	–	1:80
Hospital staff (n = 12)			
04-31	–	–	1:80
04-32	–	–	1:80
04-17	–	–	1:80
04-20	–	–	1:20

*Data not shown for patients whose samples were negative in all tests. RT-PCR, reverse-transcriptase polymerase chain reaction; Ig, immunoglobulin; –, negative result.
†RT-PCR targeting the Lassa virus glycoprotein gene (4). PCR products were detected in ethidium bromide–stained gel and sequenced (GenBank accession nos. DQ010030 and DQ010031 for 04-02 and 04-10, respectively).
‡Immunofluorescence assay used cells infected with Lassa virus strain Josiah. Findings were confirmed with μ-capture and IgG enzyme-linked immunosorbent assays (data not shown).
§Lassa virus was isolated in cell culture (strain Nig04-010), and part of the L gene was sequenced (GenBank accession no. AY693637).

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References

1. Lassa fever—Nigeria (Edo). 2004 Feb 14 [cited 2004 Dec 8]. Available from http://www.promedmail.org, archive number 20040214.0487.
2. Lassa fever, suspected—Nigeria (Edo). 2001 Mar 19 [cited 2004 Dec 8]. Available from http://www.promedmail.org, archive number 20010319.0552.
3. Demby AH, Chamberlain J, Brown DW, Clegg CS. Early diagnosis of Lassa fever by reverse transcription PCR. J Clin Microbiol. 1994;32:2898–903.
4. Drosten C, Gottig S, Schilling S, Asper M, Panning M, Schmitz H, et al. Rapid detection and quantification of RNA of Ebola and Marburg viruses, Lassa virus, Crimean-Congo hemorrhagic fever virus, Rift Valley fever virus, dengue virus, and yellow fever virus by real-time reverse transcription PCR. J Clin Microbiol. 2002;40:2323–30.
5. McCormick JB, Webb PA, Krebs JW, Johnson KM, Smith ES. A prospective study of the epidemiology and ecology of Lassa fever. J Infect Dis. 1987;155:437–44.
6. Bowen MD, Rollin PE, Ksiazek TG, Hustad HL, Bausch DG, Demby AH, et al. Genetic diversity among Lassa virus strains. J Virol. 2000;74:6992–7004.
7. Trappier SG, Conaty AL, Farrar BB, Auperin DD, McCormick JB, Fisher-Hoch SP. Evaluation of the polymerase chain reaction for diagnosis of Lassa virus infection. Am J Trop Med Hyg. 1993;49:214–21.
8. McCormick JB, King IJ, Webb PA, Scribner CL, Craven RB, Johnson KM, et al. Lassa fever. Effective therapy with ribavirin. N Engl J Med. 1986;314:20–6.

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Methicillin-resistant *Staphylococcus aureus* Skin Infections

To the Editor: Moran et al. write, "In areas with a high prevalence of CA-MRSA [community acquired methicillin-resistant *Staphylococcus aureus*], empiric treatment for skin and soft tissue infections (SSTIs) with β-lactam agents such as cephalexin may no longer be appropriate. Oral agents such as clindamycin or trimethoprim/sulfamethoxazole and rifampin should be considered in CA-MRSA" (1). However, some studies have had different results. Lee et al. reported that 31 (84%) of 37 Texas children with CA-MRSA SSTIs showed clinical improvement after incision and drainage, even though they received an "ineffective" antimicrobial agent that was not changed after the susceptibility results became available (2). These researchers also reviewed some reports with similar experience in the United States and further suggested that incision and drainage without adjunctive antimicrobial therapy were effective in immunocompetent children for CA-MRSA SSTIs <5 cm in diameter.

Several studies on Taiwanese children with CA-MRSA SSTIs agree with the viewpoint of Lee et al. Chen and colleagues reported that 22 (63%) of 35 episodes of CA-MRSA superficial soft tissue infections in children were cured by nonsusceptible antimicrobial therapy, regardless of surgical intervention (3). In a study by Wang et al., oxacillin, with or without incision and drainage, was effective in 16 (89%) of 18 children with CA-MRSA SSTIs, even in a case with high-level oxacillin resistance (MIC≥8 µg/mL) (4). Fang et al. also reported that 16 (55%) of 29 children with CA-MRSA SSTIs were eventually cured with therapy to which their infections were not susceptible (5). With these experiences and concerns about the growing problem of bacterial resistance, we suggest that incision and drainage, with or without adjunctive antimicrobial therapy, are adequate to treat noninvasive CA-MRSA SSTIs in immunocompetent children and that oxacillin or first-generation cephalosporins are still effective and sufficient under such conditions. Vancomycin and other agents that are effective against MRSA isolates should be reserved for invasive CA-MRSA infections or for immunocompromised patients. Although Moran's study was focused on adults, not on children as these studies were, we believe these suggestions are also appropriate when applied to CA-MRSA SSTIs in adults.

Finally, the antibiogram of CA-MRSA isolates may vary from country to country. In Taiwan, CA-MRSA isolates are also resistant to multiple antimicrobial agents; 71.4%, 91.4%, and 41.2% are resistant to clindamycin, erythromycin, and chloramphenicol, respectively (4). Trimethoprim/sulfamethoxazole is more effective against CA-MRSA isolates than