Vibrio cholerae O1 in 2 Coastal Villages, Papua New Guinea

To the Editor: Cholera outbreak reports are of international public health interest, especially in areas that were previously cholera free (1). Although many recent cholera outbreaks have originated in coastal areas (2), identifying the source of cholera introduction has been challenging (1). The detection of Vibrio cholerae in coastal, brackish and riverine waters in cholera-endemic and cholera-free areas supports the view that autochthonous V. cholerae is involved in the introduction of cholera (3,4). To our knowledge, cholera has not been reported in Papua New Guinea, despite social and environmental conditions likely to facilitate transmission and the nation’s close proximity to cholera-endemic countries (5,6).

On August 6, 2009, a physician who visited the coastal village of Lambutina reported an outbreak of acute watery diarrhea that was associated with the death of his father and 4 other persons from this and a neighboring village. The outbreak began in the village of Nambariwa and spread to neighboring Lambutina, Morobe Province. From August 13, multidisciplinary teams worked with the community to reduce the number of deaths through early identification and treatment of case-patients. The teams also worked to limit transmission through improvements to the water and sanitation infrastructure and by encouraging better hygiene practices among the villagers. A suspected case of cholera was defined as acute watery diarrhea or vomiting in a resident of Lambutina or Nambariwa villages since July 22, 2009. In the 2 villages, 77 cases were identified; attack rates were 14% in Lambutina (48/343) and 5.5% in Nambariwa (29/532). The overall case-fatality ratio was 6.5% (5/77); 2 patients died after they were discharged from the referral hospital.

A retrospective frequency-matched case–control study was conducted in Lambutina to identify the risk factors associated with suspected cholera. Neighborhood controls (± 5 years of age) were selected from unaffected households. Univariate and multivariate analyses were conducted with STATA version 10 (StataCorp., College Station, TX, USA).

Of the 48 case-patients in Lambutina, 43 participated in the study with 43 age-matched controls. In addition

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to having close contact with patients who had cholera, univariate analysis
showed that case-patients were more likely to have had several exposures
related to the death of other patients (Table). However, having close con-
tact with a patient was the only independent risk factor (adjusted odds ratio
4.8, 95% confidence interval 1.7–13.4) (Table). Close contact included pro-
viding nursing care for patients or carrying patients onto boats for transport
to health care facilities.

From the 10 collected samples, 4 isolates were confirmed as *V. cholerae*
O1, biotype El Tor, serotype Ogawa, by PCR detection of an O1-specific re-

gion of the *rfb* gene using established methods and PCR amplification of the
tcpA gene polymorphism specific for the El Tor biotype (7). The *ctxAB, vct*
gen genes (present in toxigenic strains) and the hemolysin gene *hlyA* (present
in all *V. cholerae* strains) were detected by PCR in all 4 isolates.

Although health authorities promptly identified and responded to the
outbreak, they could not determine its origin. The El Niño weather phe-

nomenon generates increased rainfall and elevated sea surface temperatures
and is a predictor of cholera outbreaks (8), which puts more coastal areas at
risk for such outbreaks (9). During this outbreak, Papua New Guinea re-
ported above-average rainfall (10) and warmer sea surface temperatures.
Although cholera may have been introduced to Papua New Guinea through
an infectious traveler or by other means, climatic factors may have ini-
tiated plankton blooms, the abundance of which have also been associated
with increased presence of *V. cholerae* O1. Sea and estuarine waters of these
villages are plausible sources of intro-
duction.

In Lambutina, the age-specific attack rates were lowest among young
children and increased among persons of middle age and among the elderly.
Those providing patient care and lifting during transportation as well as
those washing the bodies of the deceased may have been more represent-
ed in the >40 years age group; however, this situation may not explain the
high attack rates among the elderly.

Generally, after a cholera out-
break is detected, interventions aim to reduce the proportion of deaths to
<1%. The overall case-fatality ratio in the outbreak discussed here was 6.5%,
which reflects the challenges to ac-

cessing adequate health care in remote settings. This difficulty is exacerbated
when the disease occurs for the first
time because cholera awareness and preparedness will be weak, as can be
seen in the early management of cases during this outbreak. Villagers
who have close contact with cholera patients are at greater risk for disease
and should be a focus of interventions to limit transmission (e.g., eliminat-
ing ingestion of contaminated water, improving hygiene and sanitation).
Education to increase awareness of the disease and enhanced access to low-
osmolarity oral rehydration solution,
Hartmann solution, and zinc supple-
ments are essential.

Cholera-endemic and cholera-
nonendemic countries with coastal
populations are at an increasing risk for cholera outbreaks. Adequate prep-

aration by the health care system is vi-
tal to avoid excess deaths.

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**Alexander Rosewell,**
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**Amy Jennison,**
**Helen Smith,**
**Glen Mola,**
**Anthony Zwi,**
**and C. Raina MacIntyre**

Author affiliations: World Health Organiza-
tion, Port Moresby, Papua New Guinea (A.
Rosewell, M. Murhekar); National Depart-
ment of Health, Port Moresby (R. Dagina,
B. Ropa, E. Posanai); Port Moresby Gen-
eral Hospital, Port Moresby (S.R. Dutta); Ref-
erence Microbiology and Molecular Epidem-
ology Laboratories, Archerfield, Queens-
land, Australia (A. Jennison, H. Smith); Uni-
versity of Papua New Guinea, Port Moresby (G.
Mola); and University of New South Wales, Sydney, New South
Wales, Australia (A. Rosewell, A. Zwi, C.R.
MacIntyre).

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**Table.** Univariate and multivariate analysis of risk factors associated with suspected cholera in Lambutina village, Papua New Guinea, 2009*

| Risk factor                                      | No. cases (%) | No. controls (%) | OR (95% CI) | p value | aOR (95% CI) | p value |
|-------------------------------------------------|---------------|-----------------|------------|---------|--------------|---------|
| Attended a funeral                               | 32 (74)       | 24 (56)         | 2.3 (0.8–6.4) | 0.07    | 1.8 (0.7–4.9) | 0.25 |
| Had death in the family                          | 8 (19)        | 1 (2)           | 9.6 (1.1–214.6) | 0.02    | 2.6 (0.2–43.9) | 0.51 |
| Consumed food during funeral                     | 38 (88)       | 34 (79)         | 2.0 (0.5–7.8)  | 0.24    | NA           | NA     |
| Washed the body/clothes of deceased              | 7 (16)        | 1 (2)           | 8.2 (0.9–185.1) | 0.03    | 1.6 (0.1–28.1) | 0.74 |
| Had close contact with diarrhea patient          | 25 (58)       | 19 (9)          | 6.1 (2.1–18.3) | 0.001   | 4.8 (1.7–13.4) | 0.003 |
| Drank tap water                                  | 43 (100)      | 100 (100)       | 1.0 (NA)    | NA      | NA           | NA     |
| Boiled water for consumption                     | 1 (2)         | 0               | 1.0 (NA)    | NA      | NA           | NA     |
| Washed utensils in the ocean                     | 39 (91)       | 39 (91)         | 1.0 (0.2–5.2) | 0.64    | NA           | NA     |

*OR, odds ratio; CI, confidence interval; aOR, adjusted OR; NA, not applicable.
To the Editor: The role of clostridia as intestinal pathogens has been recognized (1). However, the full extent of the pathogenicity, clinical spectrum, and optimal therapy of Clostridium sphenoides infections remains to be determined. We describe a case of bloodstream infection in a man that was caused by C. sphenoides.

A 68-year-old man was admitted to the hospital (Harbor UCLA Medical Center, Los Angeles, CA, USA) after a motor vehicle accident in December 2009. He was afebrile (temperature 37.2°C), was hemodynamically stable, and had generalized abdominal tenderness. Computed tomography scan of the abdomen and pelvis showed laceration of the spleen and focal aortic dissection at the aortic bifurcation. The patient underwent surgical exploration and splenic resection. No signs of bowel ischemia or laceration were identified during surgery. On the fourth day postoperation, he became hypotensive, and a fever of 39.2°C developed. Blood cultures (anaerobic bottle from 1 set of blood cultures) again grew C. sphenoides. On the fourth day postoperation, he had persistent fever (38.5°C), became severely hypoxic, and was intubated. Repeat blood cultures were negative for C. sphenoides. A computed tomographic scan of the chest showed bilateral pneumonia, and a sputum culture grew Serratia marcescens. The patient underwent a 2-week course of doripenem and an 11-day course of metronidazole. He also received vancomycin for 7 days. The patient was eventually discharged to a rehabilitation facility after 2 weeks in the hospital.

C. sphenoides was initially thought not to be pathogenic in humans, but it has been occasionally reported as a human pathogen (Table 2–4). The organism is sometimes acquired from food (2). Osteomyelitis (3) and peritonitis (4) caused by C. sphenoides have also been reported. The organism has characteristic biochemical properties, and citrate is a specific substrate for the isolation of C. sphenoides (5). The pathogenesis of C. sphenoides infections in humans remains unclear. C. sphenoides may produce small alterations on Vero cells in vitro, such as turning the cells oval without altering their size, and these changes are different from those caused by C. difficile (6).

An unusual aspect of the infection in our patient was that it repre-

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