Rapid Spread of *Vibrio cholerae* O1 Throughout Kenya, 2005

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Abstract.   Between January and June 2005, 5 distinct cholera outbreaks occurred in Kenya. Overall, 990 cases and 25 deaths (2.5%) were reported. Four outbreaks occurred in towns along major highways, and 1 occurred in a refugee camp near the Sudanese border, accessible to Nairobi by daily flights. Matched case–control studies from 2 outbreaks showed that failure to treat drinking water and storing drinking water in wide-mouthed containers were significantly associated with disease. Isolates from all 5 outbreaks were *Vibrio cholerae* O1, Inaba serotype, and had genetically similar PFGE patterns of *Sfi*I-digested chromosomal DNA. Linkage of the outbreak locations by major transportation routes, their temporal proximity, and similar PFGE patterns of isolates suggests the outbreaks might have been linked epidemiologically, showing the speed and distance of cholera spread in countries like Kenya with pockets of susceptible populations connected by modern transportation. Prevention measures remain implementation of point-of-use safe water systems and case finding and referral.

INTRODUCTION

Cholera remains a disease of the world's poorest people. Since 1970 when the seventh pandemic of El Tor biotype of *Vibrio cholerae* O1 reached sub-Saharan Africa, epidemic cholera has persisted in many African countries.[1,2] While in some parts of Africa, measurable improvements in sanitation, hygiene, and overall infrastructure have occurred during recent decades, the majority of Africans still live in environments without safe drinking water or modern sanitation, putting them at ongoing risk for cholera.[2] Cholera outbreaks in Africa have been linked to multiple sources, most of these related to consumption of unsafe water and food, such as drinking river and lake water, eating at funeral feasts, and consuming food or beverages from street vendors.[5–16] Despite the multiple potential sources of cholera contamination in Africa, simultaneous countrywide or multinational epidemics, such as the explosive Latin American epidemic of the 1990s, have been rarely reported in Africa.[3,4]

Between January and June 2005, 5 discrete outbreaks of laboratory-confirmed cholera occurred throughout the country. The sites were geographically disbursed, from the Kenyan coast to the Ugandan border to the northwest corner of the country near the Sudanese border over 1,000 km away. Three outbreaks occurred along the major transnational highway connecting the Kenyan coast to Uganda. This observation, together with temporal relationship of the outbreaks, raised questions of whether these outbreaks were epidemiologically linked. We investigated these outbreaks to answer this question and to identify risk factors.

METHODS

Sites. Five cholera outbreaks in Kenya were identified by the Ministry of Health in the following locations: Busia along the Ugandan border (elevation, 1,500 m; average annual rainfall, 2,000 mm); Malindi along the Kenya's Indian Ocean coast (elevation, 20 m; average annual rainfall, 1,050 mm); Kibwezi in east-central Kenya (elevation, 1,100 m; average annual rainfall, 600 mm); Nairobi in the Eastland informal settlement (elevation, 1,800 m; average annual rainfall, 750 mm), an area with high population density and poor sanitary services of mixed ethnicity, but notably includes refugee population of Sudanese and Somalis; and Kakuma refugee camp and the adjacent town in northeast Kenya (elevation, 0–250 m; annual rainfall, less than 250 mm), 80 miles by road from the Sudanese border (Figure 1). In most of Kenya, January–February are dry months, followed by a rainy season from March to June. Kakuma is semi-arid year-round.

Case definition and case finding. A case of suspected cholera was defined as any person of any age with profuse, effortless watery diarrhea with 3 or more stools in 24 hours, residing in the affected areas, defined as the entire district for Busia and Malindi, Kibwezi division, Eastland area in Nairobi, and Kakuma refugee camp and surrounding areas, occurring during an interval defined as follows: January 1–March 31, 2005, in Busia; February 15–March 31, 2005, in Malindi; February 1–March 31, 2005, in Kibwezi; June 1–20, 2005, in Nairobi; and November 1, 2004–June 30, 2005 in Kakuma. Only persons admitted to the hospital were included in the Malindi, Kakuma, and Kibwezi investigations. In Busia and Nairobi, both inpatients and outpatients were included. Case finding was done through review of hospital and outpatient facilities in the affected areas by local, district, and national Ministry of Health personnel.

Risk-factor analysis. Case-control studies to evaluate risk factors for infection were conducted in Malindi and Kibwezi. An additional case-control study was done in Kakuma; because the epidemiology of refugee camps is distinct, the results of that case-control study will be presented elsewhere. In Malindi, all cases were culture-confirmed. In Kibwezi, cases were culture-confirmed or epidemiologically linked to a culture-confirmed case, defined as meeting the clinical case definition and living in the same homestead as a culture-confirmed case. Controls were neighborhood- and age-matched to cases and did not report having a diarrheal illness during the outbreak period. Two controls were selected for each case through systematic random sampling by spinning a bottle at the case's house and walking to the third house in

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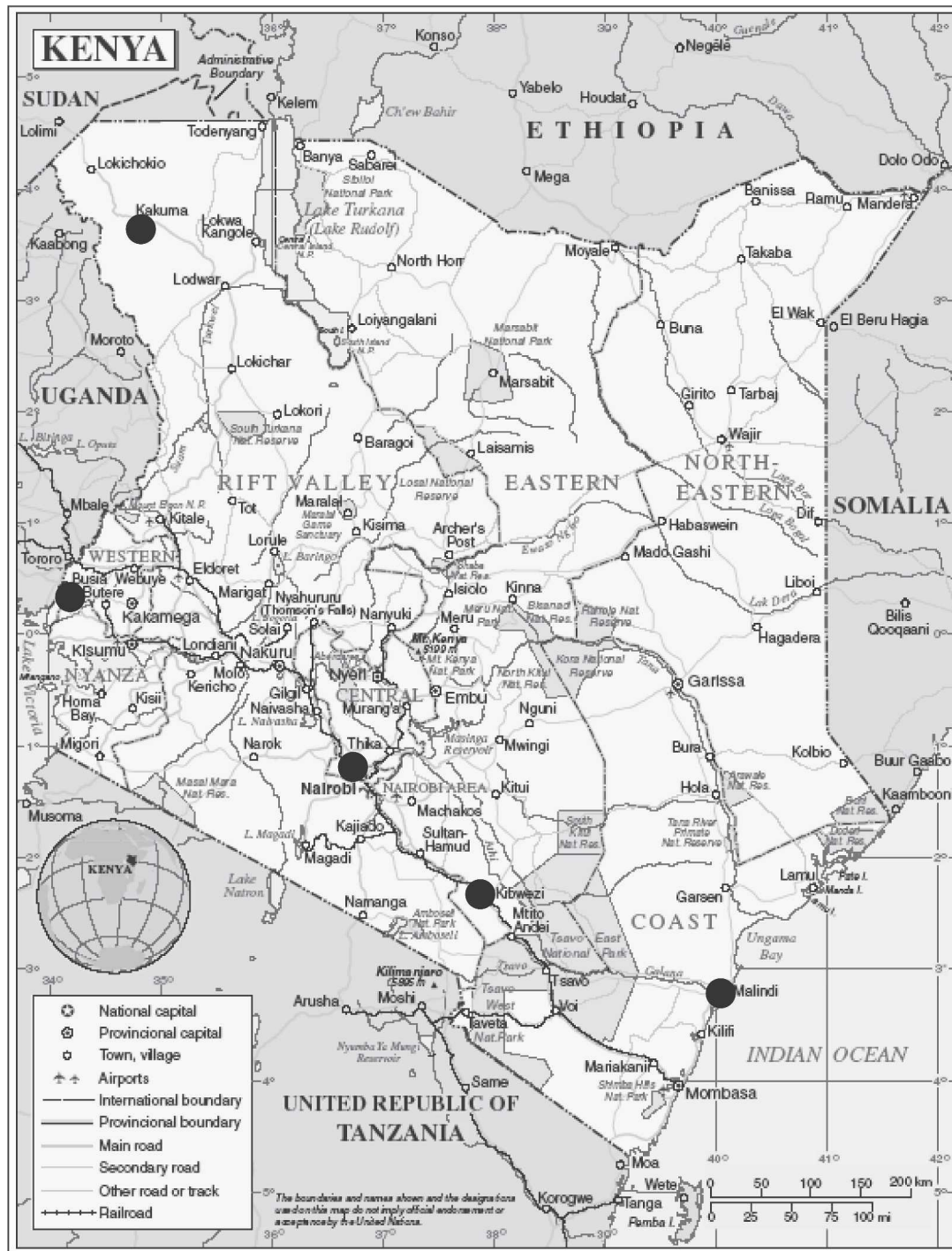
FIGURE 1.   Map of Kenya with sites of the 5 outbreaks indicated by large dots.

that direction to find an appropriate age-matched control. If no appropriate control was found in that house, the next house in the same direction was visited, and so on, until an appropriate control was enrolled. The search for the second control followed the same procedure starting at the first control's house and spinning the bottle again. Controls were age-matched according to the defined age categories: in Malindi, 0–5, 6–15, 16–35, and > 35 years old, and in Kibwezi, 0–4, 5–14, 15–20, 21–30, 31–40, 41–50, and > 50 years old. Standardized questionnaires were administered in both areas, al-

though some questions on foods and activities varied by locality. Potential risk factors for cases and matched controls were asked about in the 5-day period before the onset of symptoms of the case. Risk factors assessed were hygienic practices (e.g., hand-washing, latrine use), travel history, food history, water sources and handling practices, contact with possible cases of cholera, and attendance at funerals.

Data entry and analysis was done using EpiInfo, version 3.3.2 (CDC, Atlanta, GA). Univariate analysis of categorical variables was performed, and adjusted odds ratios were cal-

culated using the Mantel–Haenszel test, stratifying by case-control triplets. All variables with $P \leq 0.1$ in univariate analysis were included in multivariable conditional logistic regression models, and a backward elimination procedure was used. Interaction of significant variables was assessed by inclusion of product terms in the model, using a P value of 0.05 as the determinant of significant interaction. Because of clustering of cases within households, unconditional logistic regression of the case-control studies that controlled for clustering was performed using the generalizing estimating equation (PROC GENMOD in SAS for Windows, version 9.1; SAS Institute, Inc., Cary, NC).

Laboratory testing. Primary microbiological assessment of stool was done at several laboratories in Kenya, at the Busia District Hospital, Malindi District Hospital, Makindu sub-District Hospital, the National Public Health Laboratory in Nairobi, and the KEMRI/CDC Laboratory in Kisian, near Kisumu. Stool was either directly plated on thiosulfate–citrate–bile salts agar (TCBS) or transported on Cary–Blair transport media and then plated on TCBS agar. Colonies of growth were evaluated using standard biochemical reactions, and *V. cholerae*-positive isolates were serogrouped and serotyped using agglutination tests with commercial antisera.[17]

Cholera isolates that had been saved were sent to the Kenya Medical Research Institute (KEMRI), Center for Microbiology Research, in Nairobi. These isolates underwent antimicrobial susceptibility testing by the disk diffusion method using standardized thresholds for defining susceptibility.[18] *Sfi*I-digested chromosomal DNA was prepared in agarose plugs using a 1-day (24–28 h) protocol for molecular subtyping of *V. cholerae,* as described, with minor modifications.[19] Pulsed-field gel electrophoresis (PFGE) was performed with a CHEF DRIII system (Bio-Rad Laboratories, Richmond, CA) on a horizontal 1% agarose gel for 14 h at 90 V, with a pulse time of 2–8 s at 14°C. The restriction endonuclease digest patterns were compared by the method of Tenover.[20] For comparison of PFGE patterns in the region, *V. cholerae* isolates from Uganda and South Sudan from January to September 2005 were also sent by the Ugandan Central Public Health Laboratory to KEMRI.

RESULTS

Descriptive epidemiology. Overall, 990 suspected cholera cases, including 186 (18.8%) laboratory-confirmed cases,

were reported between January and June 2005 from these 5 outbreaks in Kenya (Table 1); 25 deaths were documented (minimum case-fatality ratio = 2.5%). Children and adolescents accounted for half of the patients; 20% were under 5 years of age, and 31% were of age 5–17 years. The 5 outbreaks occurred in discrete clusters, each with a single peak (Figure 2). The outbreaks were fairly widely dispersed (geographically) within each location. In Busia, cases resided in multiple villages within 3 large geopolitical areas, called Divisions. In Kibwezi, 63% of cases came from 3 adjacent villages, and the remainder of cases came from 29 other villages. In Malindi, over half of the cases came from 1 village. In Nairobi, 51 (70%) cases resided in Motherlands area with the rest of the cases residing in 7 different areas. The Kakuma outbreak occurred predominantly within the camp, with some spillover into the adjacent town. The cases in each of the outbreaks did not differ significantly by sex or age distribution from the total number of cases in the 5 outbreaks.

Case-control studies. In both case-control studies, 34 cases and 68 controls were enrolled. Cases and controls did not differ significantly by sex. In Kibwezi, of the 3 risk factors found to be significant in univariate analysis, 2 remained significant in multivariable analysis: not treating drinking water (OR 4.9, 95% CI 1.6–15) and observation of storage of water in a bucket in the home (OR 3.8, 95% CI 1.2–12) (Table 2). In Malindi, 9 risk factors were significantly associated with being a case in univariate analysis (Table 3). Of these, 3 were significant in multivariable analysis: attending a funeral (OR 51, 95% CI 4.7–560), eating ugali, a maize meal staple, outside the home (OR 6.8, 95% CI 1.3–35), and storing water in an open container (OR 3.3, 95% CI 1.0–10). Of the 25 cases that reported attending a funeral in the 5 days before illness, 18 (73%) attended 1 funeral in the village where the majority of cases occurred. In addition, 23 (92%) of cases who attended a funeral reported eating at the funeral whereas only 2 (12%) of controls who attended a funeral reported eating there ($P <$ 0.001). In neither outbreak was the source of drinking water implicated. In Kibwezi, 62% of cases used river water and 27% used irrigation canal water, compared with 66% and 18% of controls, respectively. In Malindi, most cases (88%) and controls (98%) used drinking water purchased from a communal tap.

Laboratory results. *V. cholerae* O1 was isolated from 186 stools (Table 1). All isolates were of serotype Inaba. Antimicrobial susceptibility testing was done on 40 isolates from

TABLE 1
Descriptive epidemiology of 5 cholera outbreaks in Kenya, January–July 2005

Characteristic	Busia	Malindi	Kibwezi	Nairobi	Kakuma	Total
			Location			
No. of suspected cases	164	58	173	73	522	990
Inpatients	41	58	173	25	522	819
Outpatients	123	0	0	48	0	171
No. of laboratory-confirmed cases (%)	63 (38.4)	26 (45)	33 (19.1)	20 (27.4)	44 (8.4)	186 (18.8)
Male sex, n (%)	63 (38.4)	26 (44.9)	93 (54)	36 (49)	264 (51)	482 (48.7)
Age groups, n (%)						
0–4 years	32 (20)	9 (15.5)	55 (32)	8 (11)	87 (17)	191 (20)
5–17	43 (26)	20 (34.9)	50 (29)	19 (26)	168 (32)	300 (31)
18–35	59 (36)	24 (41.5)	41 (24)	35 (48)	174 (33)	333 (34)
36–49	9 (6)	4 (6.9)	9 (5)	10 (14)	41 (8)	73 (7)
≥ 50	21 (13)	1 (1.7)	6* (3)	1 (1)	32* (6)	61 (6)
No. of deaths (case-fatality ratio)	8 (4.8)	0	2 (1.2)	1 (1.4)	14 (2.7)	25 (2.5)

* Numbers for age not equal to total because age information was lacking for some patients.
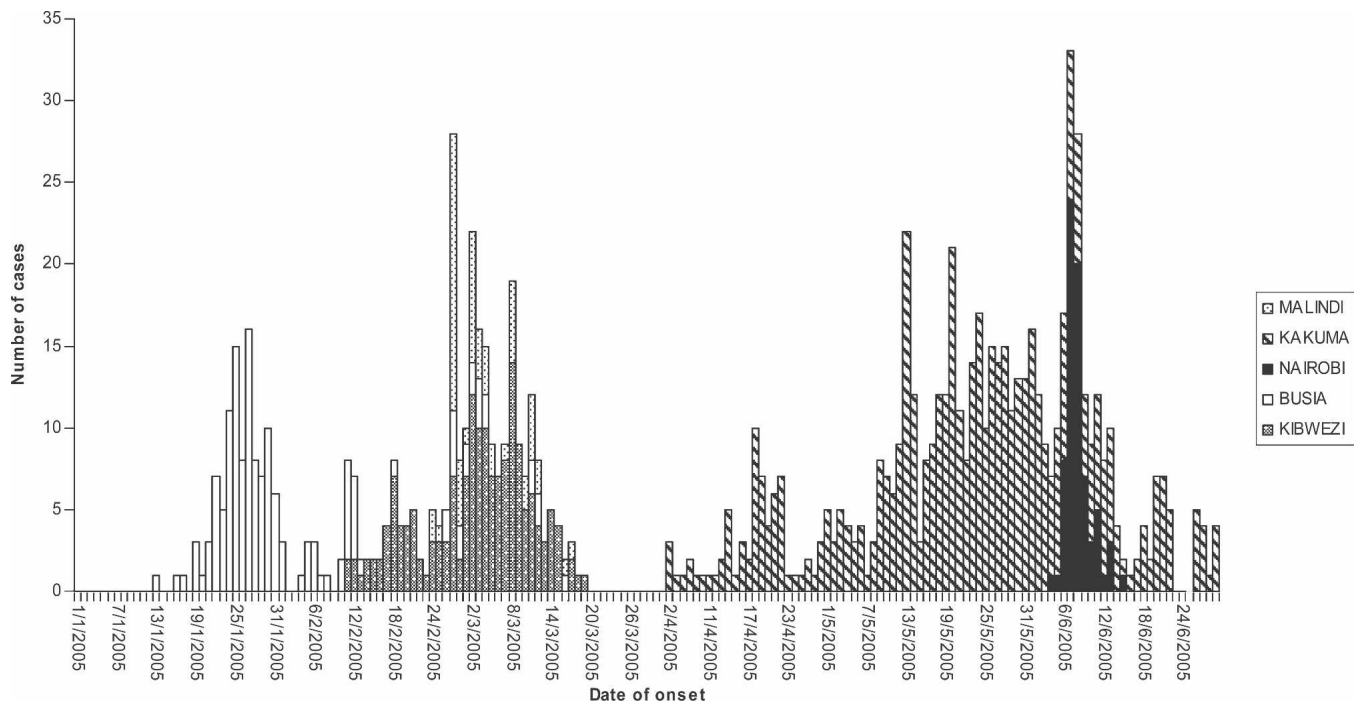
FIGURE 2.   Epidemic curve of 5 cholera outbreaks in Kenya, January–June 2005

each of the 5 outbreaks. Of these, 39 were susceptible to tetracycline (1 exhibited intermediate resistance). Thirty-one isolates had similar antimicrobial susceptibility patterns, and 5 others only differed in susceptibility to 1 antibiotic. PFGE of *Sfi*I-digested chromosomal DNA, done on 16 Kenyan isolates, indicated that isolates from all 5 of these outbreaks were genetically similar, differing by fewer than 3 bands[20] (Figure 3). In addition, 6 isolates from outbreaks in Uganda, including 1 near the Ugandan border and 1 near the South Sudan border, and 2 isolates from an outbreak in South Sudan showed similar PFGE patterns with the Kenyan isolates (Figure 3).

DISCUSSION

In the first half of 2005, Kenya experienced 5 cholera outbreaks with nearly 1,000 reported suspect cases in geographically discrete locations over 1,000 km apart. PFGE of *Sfi*I-digested chromosomal DNA showed that the isolates causing these outbreaks were genetically similar. In addition, outbreaks in neighboring Uganda and Sudan during the same time period were caused by genetically similar strains. This genetic similarity, along with the temporal clustering and transportation connections between the sites, suggests that some or all of these outbreaks might have been linked epidemiologically, showing that cholera can rapidly spread across a wide geographic area in modern-day Africa.

The reason why these outbreaks occurred in early 2005 is not clear. Climatic conditions were not unusual in early 2005. Cholera outbreaks are associated with crowded living conditions, inadequate or unprotected water supply, and poor sanitation, which exist in much of Kenya, making most of the country susceptible to outbreaks if *V. cholerae* are introduced.[21] In addition, Kenya has 2 large refugee camps, which are known to be fertile ground for explosive cholera out-

breaks.[8,15] Although the socioeconomic and sanitary conditions capable of supporting cholera outbreaks existed in much of Kenya, the nearly simultaneous occurrence of several outbreaks in early 2005 in distant locations likely required another important factor: an active transportation infrastructure. Many large outbreaks of cholera in Africa have been described before, although most of these occurred in 1 location.[5–16] Spread throughout an African country has been rarely reported. In 1992, cholera moved southward along the shores of Lake Tanganyika in Burundi, and in 1997, cholera spread in western Kenya along the shores of Lake Victoria.[6,13] In both of these situations, however, the spread was along contiguous districts bordering lakes, whose water was found to harbor cholera.

The possibility of rapid spread over geographically distant areas described here is similar to circumstances in Peru in 1991, where an outbreak in a coastal city subsequently disseminated throughout Peru and other countries in Latin America over the course of the next year.[1,2] Presumably, the capacity for cholera to spread more rapidly in Latin America than in Africa was that, in general, Latin American transpor-

TABLE 2
Significant risk factors for cholera in the Kibwezi outbreak, Kenya, 2005

Risk factor	Cases N (%)	Controls N (%)	Univariate OR (95% CI)	Multivariate OR (95% CI)
Not treating drinking water	25 (73.5)	31 (45.6)	3.4 (1.3–8.9)	4.9 (1.6–15)
Storing water in a bucket*	19 (55.9)	23 (33.8)	2.9 (1.2–7.5)	3.8 (1.2–12)
Unsound latrine superstructure†	22 (64.7)	32 (47.1)	4.0 (1.1–14)	NS‡

* Observed by the interviewer.
† Defined as "walls did not provide enough privacy for an adult during the day."
‡ Not significant.

TABLE 3
Significant risk factors for cholera in the Malindi outbreak, Kenya, 2005

Risk factors	Cases N (%)	Controls N (%)	Univariate OR (95% CI)	Multivariate OR (95% CI)
Attended funeral	25 (74)	17 (25)	34.0 (4.0–291)	51 (4.7–560)
Eating ugali outside the home	9 (27)	6 (9)	3.0 (1.0–8.7)	6.8 (1.3–35)
Storing drinking water in open container	19 (55)	14 (21)	4.4 (1.7–11)	3.3 (1.0–10)
Eating beans outside the home	10 (29)	7 (10)	2.9 (1.1–7.5)	NS*
Changing main source of drinking water	14 (41)	7 (10)	4.5 (1.6–12)	NS
Not treating drinking water	30 (88)	44 (65)	5.0 (1.4–18)	NS
Drinking water from outside the home	25 (76)	25 (38)	4.1 (1.7–9.8)	NS
Not washing hands with soap before eating	25 (74)	34 (52)	3.7 (1.3–10.3)	NS
Not washing hands after visiting toilet	12 (38)	10 (16)	4.3 (1.2–15)	NS

* Not significant.

tation infrastructure was much more developed, facilitating ease and speed of long-distance spread of the bacteria by infected people. Kenya's transportation sector has improved over the last decade. Three towns with outbreaks (Kibwezi, Nairobi, and Busia) are all stopovers on the major highway connecting the Kenyan coast on the Indian Ocean to the Uganda border. Malindi is 100 km along a well-traveled tarmac road north of Mombasa, the eastern terminus of the transnational highway. This transnational highway is the most heavily used thoroughfare in the country, and a vehicle can cross the entire country in a day on this highway. Kakuma is not along this highway, but because it has a major refugee camp, there is daily transport between Kakuma and Nairobi by both road and plane. The slum area in Nairobi where the outbreak occurred is a residence of families of many refugees from Kakuma, and there is substantial mobility from the refugee camp to this community. Moreover, 1 of the Ugandan outbreaks, in Arua in North Uganda, is another area where Sudanese refugees reside. Thus Arua, as well as the South Sudan outbreak, might also have been linked to the Kenyan outbreaks through migration of infected persons. Transportation routes in Africa have been implicated in other infectious disease epidemics, most notably HIV.[22] As the transportation infrastructure modernizes ahead of other types of infrastructure (like water quality and sewage management) in African countries, the dissemination of diseases of underdevelopment, like cholera, might be enhanced.

The genetic similarity of the cholera isolates from these outbreaks does not necessarily prove an epidemiologic link but rather could reflect a predominant clonal strain in circulation in Kenya and East Africa during this time period. Previous studies in V. cholerae showed that similar PFGE patterns of isolates tend to occur in outbreaks, such as the Latin American outbreak of the 1990s, or with stable, relatively isolated environmental reservoirs, such as in the U.S. Gulf Coast or Australia.[23] Clonal PFGE patterns of V. cholerae have also been seen before from multiple African countries over a several-year period.[24] If not epidemiologically linked, the temporal clustering of outbreaks of cholera in East Africa in 2005 suggests that multiple foci of genetically similar strains of cholera exist in local environmental reservoirs, which, under the right circumstances, can erupt into multiple outbreaks in a region simultaneously.

No clear single source or route of transmission for these outbreaks was identified. Similarly, in the Latin American experience, no single source caused all the outbreak foci.[1,2] Contaminated water, either at the source or in the home, was likely a source of infection in the Kenya outbreaks, based on

findings during past outbreaks in Africa and on the results of our 2 case-control studies.[6–9,11,13,16] Two main risk factors identified in our case-control studies were not treating drinking water at the point of use and storing water in widemouthed containers. Water contaminated during storage has been associated with cholera in other parts of the world.[7,8,25] Stored water usually has been found to have a higher level of contamination than source waters.[25,26] This is because water stored in wide-mouthed containers is more likely to be contaminated due to introduction of unclean hands and contaminated utensils.[26] Contamination of stored water can be prevented by safe water systems that include point-of-use treatment of water with sodium hypochlorite and safe water storage in containers with a narrow mouth, lid, and a spigot to prevent recontamination and encourage behavioral changes.[26,27] Point-of-use safe water systems have been shown to be protective during cholera outbreaks in Madagascar and Latin America.[4,16]

In addition to water-related risk factors, funeral attendance is another known risk factor for cholera in Africa, either through eating communal food or touching the body.[6,10,12] We found that funeral attendance in Malindi was a risk factor. Of those who said they attended a funeral, three-quarters attended a funeral in 1 village where the majority of cases occurred. Eating at the funeral was shown to be a risk factor
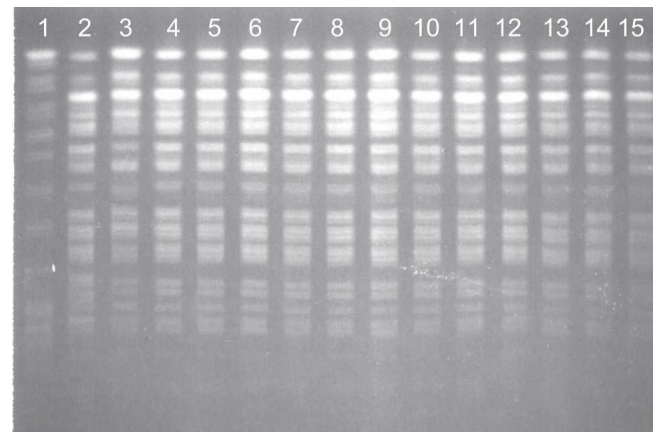

FIGURE 3.   PFGE of SfiI-digested chromosomal DNA from Vibrio cholerae isolates from 5 outbreaks in Kenya, 2 outbreaks in Uganda, and 1 in South Sudan, 2005. Lane 1, size marker Salmonella enterica serotype Braenderup H9812 (XbaI-digested); lane 2, Malindi; lanes 3 and 4, Busia; lanes 5 and 6, Kibwezi; lanes 7 and 8, Nairobi; lanes 9 and 10, Kakuma; lane 11, Arua, northern Uganda; lanes 12 and 13, Bundibugyo, western Uganda; lanes 14 and 15, South Sudan.

for illness. Although some individuals anecdotally reported touching the body, we did not ask about touching or washing the body in the case-control study questionnaire. Of note, the funeral was for a woman with suspected cholera who never presented at a health facility. This highlights the point that mortality from cholera is higher in those who do not seek medical care, making active case finding and referral an important intervention in cholera outbreaks. The case-fatality ratio of 2.5% seen in the Kenya outbreaks is higher than in many other cholera outbreaks and suggests the need for earlier referral and better case management.[5–16]

In Malindi, eating ugali (maize meal) outside the home was a risk factor, although only 27% of cases had this exposure. We could not distinguish if this represented exposure to a particular contaminated product, a particular restaurant, or if it served as an indicator of eating other potentially contaminated foods outside the home. Particular food items, such as millet, peanut sauce, rice, cooked peas, and raw vegetables, have been associated with cholera before, suggesting that many types of food can be vehicles of cholera in the setting of an outbreak.[5,8–10,14] The association of cholera with eating at the funeral and with ugali highlights that, in cholera outbreaks, safe water interventions alone might not be enough to prevent transmission, and reducing exposure to potentially contaminated food sources is also important.

Our investigation reinforces several important public health points regarding cholera in Africa. As previously emphasized, preventive measures in outbreaks of water-borne diseases, like cholera, should include point-of-use safe water interventions, including treatment and safe storage.[16,23] Emphasis on point-of-use water interventions, eating safe foods, and active case finding and referral should not be sidelined by attempts to identify and rectify the source of contamination. Our investigation also emphasizes that spread of food- and water-borne diseases can occur rapidly and widely in countries like Kenya that have areas of poor sanitation and unsafe water supply, which can be fertile ground for diseases that can be transported within days or even hours by road, rail, or air. Public health officials should be aware of the potential for such rapid spread and thereby target health-communication messages and preventive strategies widely.

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