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Description of a Mass Poisoning in a Rural District in Mozambique: The First Documented Bongkrekic Acid Poisoning in Africa

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Background. On 9 January 2015, in a rural town in Mozambique, >230 persons became sick and 75 died of an illness linked to drinking *pombe*, a traditional alcoholic beverage.

Methods. An investigation was conducted to identify case patients and determine the cause of the outbreak. A case patient was defined as any resident of Chitima who developed any new or unexplained neurologic, gastrointestinal, or cardiovascular symptom from 9 January at 6:00 AM through 12 January at 11:59 PM. We conducted medical record reviews, healthcare worker and community surveys, anthropologic and toxicologic investigations of local medicinal plants and commercial pesticides, and laboratory testing of the suspect and control *pombe*.

Results. We identified 234 case patients; 75 (32%) died and 159 recovered. Overall, 61% of case patients were female (n = 142), and ages ranged from 1 to 87 years (median, 30 years). Signs and symptoms included abdominal pain, diarrhea, vomiting, and generalized malaise. Death was preceded by psychomotor agitation and abnormal posturing. The median interval from *pombe* consumption to symptom onset was 16 hours. Toxic levels of bongkrekic acid (BA) were detected in the suspect *pombe* but not the control *pombe*. *Burkholderia gladioli* pathovar *cocovenenans*, the bacteria that produces BA, was detected in the flour used to make the *pombe*.

Conclusions. We report for the first time an outbreak of a highly lethal illness linked to BA, a deadly food-borne toxin in Africa. Given that no previous outbreaks have been recognized outside Asia, our investigation suggests that BA might be an unrecognized cause of toxic outbreaks globally.

Keywords. Bongkrekic acid; *Burkholderia gladioli*; foodborne; outbreak; poisoning.

On 9 January 2015 at approximately 10:00 PM, 4 patients went to the Chitima Health Center in Tete Province, Mozambique, after experiencing sudden weakness, abdominal pain, and diarrhea. Overnight, 47 additional patients presented with similar complaints. During the following 96 hours, a total of 234 persons became sick, of whom 75 died. Affected individuals reported attending a funeral ceremony on 9 January and drinking *pombe*, a locally brewed alcoholic beverage made from corn flour.

METHODS

Outbreak Setting

Chitima is a rural community of 20 135 inhabitants [1], located in Tete Province, in northwestern Mozambique. During the outbreak, most affected patients presented to the Chitima Health Center, the primary healthcare facility in the village. Once capacity was exceeded at the Chitima Health Center, some patients were transferred to the Rural Hospital in Songo, approximately 30 km away (Figure 1). The Tete Provincial Health Department reported the outbreak to the Mozambique Ministry of Health, which dispatched an investigation team of clinicians and public health officials to Chitima on 12 January.

Epidemiologic Investigation

A multidisciplinary team comprising representatives from the Mozambique Ministry of Health, the Tete Provincial Health Department, the US Centers for Disease Control and Prevention (CDC), and the World Health Organization went to Chitima to investigate the outbreak. A case patient was defined as any Chitima resident who developed any new or unexplained neurologic, gastrointestinal, or cardiovascular symptom (Table 1) from 9 January at 6:00 AM through 12 January at 11:59 PM.

We identified potential case patients by (1) reviewing all of the medical records of patients presenting during January 2015

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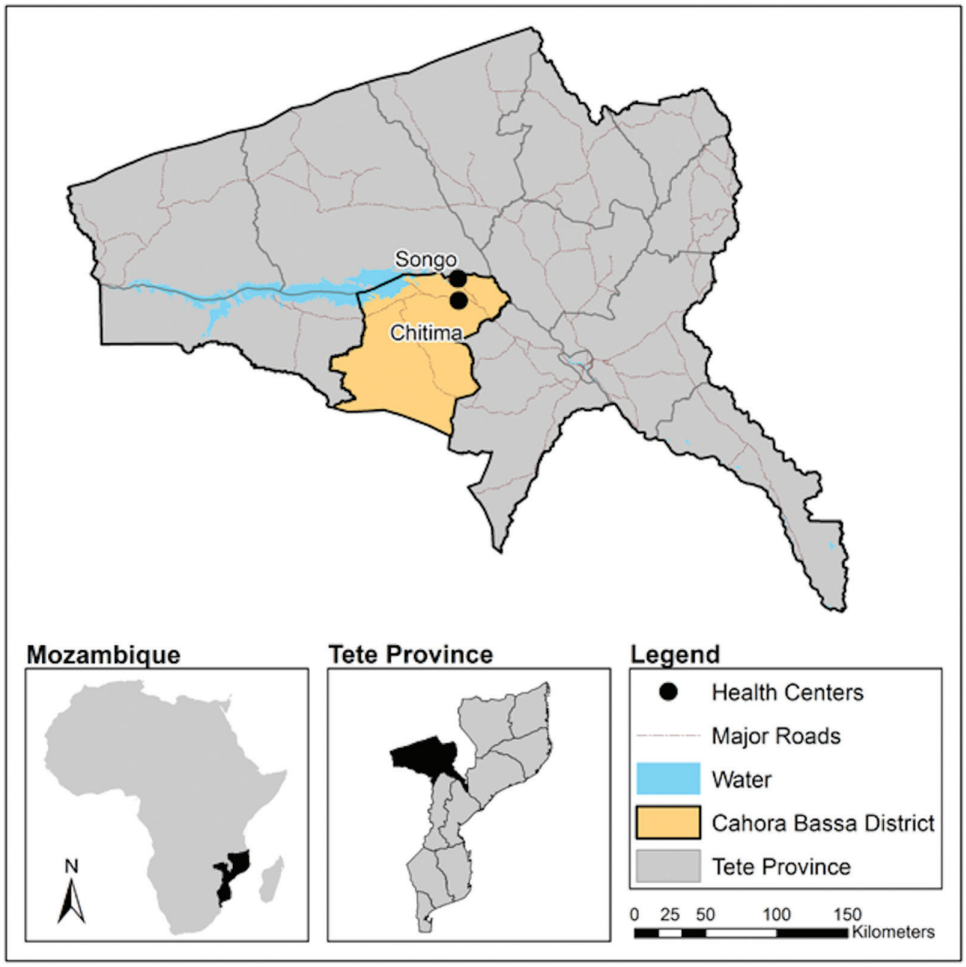

Figure 1. Location of outbreak: Cahora Bassa District, Tete Province, Mozambique.

and interviewing clinicians at the Chitima Health Center and Songo Rural Hospital, (2) interviewing hospitalized patients, and (3) soliciting from local community leaders lists of persons who had become sick or died. Deaths were confirmed with healthcare providers and family members of the deceased.

We interviewed affected individuals during 13–19 January 2015. After obtaining verbal informed consent, we used a standardized questionnaire to collect data regarding symptoms and food and beverage consumption in the 48 hours before symptom onset. Family members were interviewed as proxies for deceased individuals. Interviewers provided a series of commonly used cups of known volume to assist individuals in estimating the quantity of *pombe* consumed. Ultimately, some individuals who were interviewed did not meet the case definition; these individuals were chosen as a convenience sample of noncase patients to compare with case patients regarding potential exposures.

To assess any continuing sequelae 4 weeks after the poisoning event, during 5–7 February 2015 the team returned to Chitima to interview and examine 17 survivors identified by community leaders. During this visit, the team conducted a toxicologic field

Table 1. Symptoms Included in Case Definition[a]

Symptom
Neurologic
Agitation
Confusion
Headache
Vertigo
Loss of consciousness
Weakness
Lethargy
Convulsions
Paresthesia
Hallucinations
Gastrointestinal
Abdominal pain
Nausea
Vomiting
Diarrhea
Cardiovascular
Chest pain
Palpitations

[a]Case patients were defined as any Chitima residents who experienced any new or unexplained symptom from this list from 9 January 2015 at 6:00 AM through 12 January 2015 at 11:59 PM.

investigation by visiting the Chitima District Office of Economic Activities and vendors at informal markets to identify locally available toxic chemicals, conducted a literature review to identify endemic toxic plants, and consulted local traditional healers to discuss local home remedies and poisons. We also looked for potential contaminants in the house where the corn flour used to make the *pombe* was stored and at all places where the *pombe* was prepared and sold.

Laboratory Investigation

Forensic pathologists performed autopsies on 2 deceased patients. In addition to gross examination, they obtained tissue specimens of brain, spleen, stomach, small and large intestines, lung, heart, liver, pancreas, and kidney. Tissue samples were preserved in formaldehyde and shipped to the national pathology center for slide preparation, staining, and microscopic examination.

Local officials collected samples of the suspect *pombe* and corn flour on 10 January; the samples were transported to the Instituto Nacional de Saúde laboratory in Maputo and frozen at −20°C. We collected a sample of recently prepared *pombe* from a neighboring town as a control for laboratory analysis. *Pombe* samples were sent to a private laboratory in Mozambique and laboratories in Portugal, South Africa, and the United States. Samples were tested for a variety of toxic and biologic substances, including pesticides, heavy metals, volatile compounds, cyanide, and methanol. The US Food and Drug Administration (FDA) Forensic Chemistry Center laboratory expanded testing of the *pombe* and corn flour samples using a battery of forensic screening tests. These tests included nontargeted and quantitative liquid chromatography–mass spectrometry and microbiologic testing [2].

Statistical Analysis

Using Epi Info (version 7.1.3.10; CDC, 2012) and SAS 9.3 (SAS Institute) software, we analyzed potential associations between demographic factors and food and beverage consumption with illness and death, using the χ^2 and Fisher exact tests for categorical variables. Severity of illness was categorized as recovered, not hospitalized; recovered, hospitalized; or death. For continuous variables, we performed *t* tests for normally distributed and Wilcoxon rank sum tests for nonnormally distributed variables. We used the Kruskal-Wallis test to evaluate the dose-response relationship between the amount of *pombe* consumed and clinical outcomes. Alpha values ≤.05 were considered statistically significant.

RESULTS

Epidemiologic Investigation

We identified 234 persons who met the case definition. Overall, 61% of the persons identified as case patients were female (n = 142) (Table 2). Age ranged from 1 to 87 years, with a median age of 30 years. Increasing age was associated with death (P = .003). Affected individuals resided in all 10 neighborhoods of Chitima; however, the majority (n = 111) lived in the Cawira B neighborhood, where the *pombe* was both prepared and sold. Of the 234 case patients, 103 were hospitalized and 75 died (case fatality rate, 32.1%). Fifty-one (68.0%) of those who died had been hospitalized; the remaining 24 (32.0%) did not seek medical attention, probably because they

Table 2. Demographic Characteristics and Clinical Manifestations in 234 Chitima Residents With Disease Onset During 9–12 January 2015

Characteristic or Manifestation	Case Patients by Outcome			
	Recovered, Not Hospitalized (n = 107)	Recovered, Hospitalized (n = 52)	Death (n = 75)	All Case Patients (n = 234)
Sex, No. (%)				
Male	38 (36)	21 (40)	33 (44)	92 (39)
Female	69 (64)	31 (60)	42 (56)	142 (61)
Age, median (range), y[a]	29 (1–87)	32 (6–80)	39 (3–78)	30 (1–87)
Age unknown, No.	4 (4)	4 (8)	19 (25)	27 (12)
Signs and symptoms, No (%)	n = 107	n = 52	n = 46	n = 205
Abdominal pain	70 (65)	39 (75)	27 (59)	136 (66)
Diarrhea	65 (61)	35 (67)	25 (54)	125 (61)
Vomiting	49 (46)	33 (63)	34 (74)	116 (57)
Weakness	45 (42)	30 (58)	26 (56)	101 (49)
Palpitations	28 (26)	22 (42)	19 (41)	69 (34)
Chest pain	26 (24)	11 (21)	16 (35)	53 (26)
Vertigo	22 (21)	20 (38)	5 (11)	47 (23)
Headache	27 (25)	11 (21)	6 (13)	44 (21)
Nausea	22 (21)	9 (17)	12 (26)	43 (21)
Dyspnea	5 (5)	1 (2)	13 (28)	19 (9)
Psychomotor agitation	2 (2)	0 (0)	14 (30)	16 (8)

[a]Differences in mean age among the 3 outcome groups were statistically significant (P = .01; Kruskal-Wallis test).

were too ill to walk to a health facility. The earliest reported symptom onset was 4:00 PM on 9 January. The first recorded death was overnight on 9 January, and the last recorded death occurred on 16 January (Figure 2).

Data regarding the time of *pombe* consumption and time of symptom onset were available for 120 patients (51.3%). The median time to symptom onset was 16 hours after *pombe* consumption (range, 0–148 hours). Clinical data were available for 205 individuals (87.6%). The most common initial signs and symptoms were abdominal pain (n = 136; 66.3%), diarrhea (n = 125; 61.0%), and vomiting (n = 116; 56.6%), followed by nonspecific neurologic symptoms, including generalized malaise (n = 101; 49.2%), vertigo (n = 47; 22.9%), and headache (n = 44; 21.5%) (Table 2). Chest pain was also a common symptom (n = 53; 25.9%).

Initial vital signs were generally unremarkable. No hypersalivation, diaphoresis, or miosis were noted. Patients who died developed progressive confusion, followed by loss of consciousness, abnormal posturing, and death. Some gravely ill patients had rigidity at examination and opisthotonic posturing, but no ataxia, tremor, cranial nerve abnormalities, or tonic-clonic seizures. Clinical laboratory findings from 45 patients who died are listed in Supplementary Table S1. Four weeks later, 13 of the 17 interviewed survivors reported ongoing symptoms, including neurologic signs and symptoms (eg, headache, distal extremity paresthesia, weakness [n = 12; 70.6%] and palpitations [n = 7; 41.2%]). No deficits in proprioception were noted. In general, gastrointestinal signs and symptoms resolved without specific treatment.

Data regarding food or beverage consumption in the 48 hours before the outbreak were available for 276 individuals, including all 234 case patients (Table 3). Of those meeting the case definition, 232 (99.1%) reported drinking *pombe*. *Xima*, a staple food made from corn flour, was consumed by 151 (64.5%); however, there was no common source, and the *xima* was generally prepared and eaten at home.

Of the 267 individuals who consumed *pombe*, 232 developed illness meeting the case definition. Compared with the 42 persons who were not case patients, those who were case patients had greatly increased odds of consuming *pombe* (odds ratio, 23.2; 95% confidence interval, 4.6–116.2). Only 2 individuals meeting the case definition denied consuming *pombe*. Neither was gravely ill, and both had only a single qualifying symptom (headache or abdominal pain). Those who died drank more *pombe* per body weight than those who survived (median, 7.9 vs 4.5 mL/kg; $P = .001$) (Table 3). Those who died had a shorter interval between drinking *pombe* and disease onset than survivors (12 vs 17 hours; $P = .004$).

We interviewed 92 individuals about the characteristics of the *pombe*. Most respondents did not report any unusual taste or odor of the *pombe* (n = 56; 60.9%); no particular abnormality was reported consistently among those who did report unusual taste and odor (Supplementary Table S2).

The woman who prepared the *pombe* was one of the first casualties of the outbreak. She prepared the *pombe* in her home before delivering it to a nearby kiosk at 6:00 AM to be sold at the funeral ceremony; she was not seen again before being found dead in her home at 2:00 AM the following morning. The corn

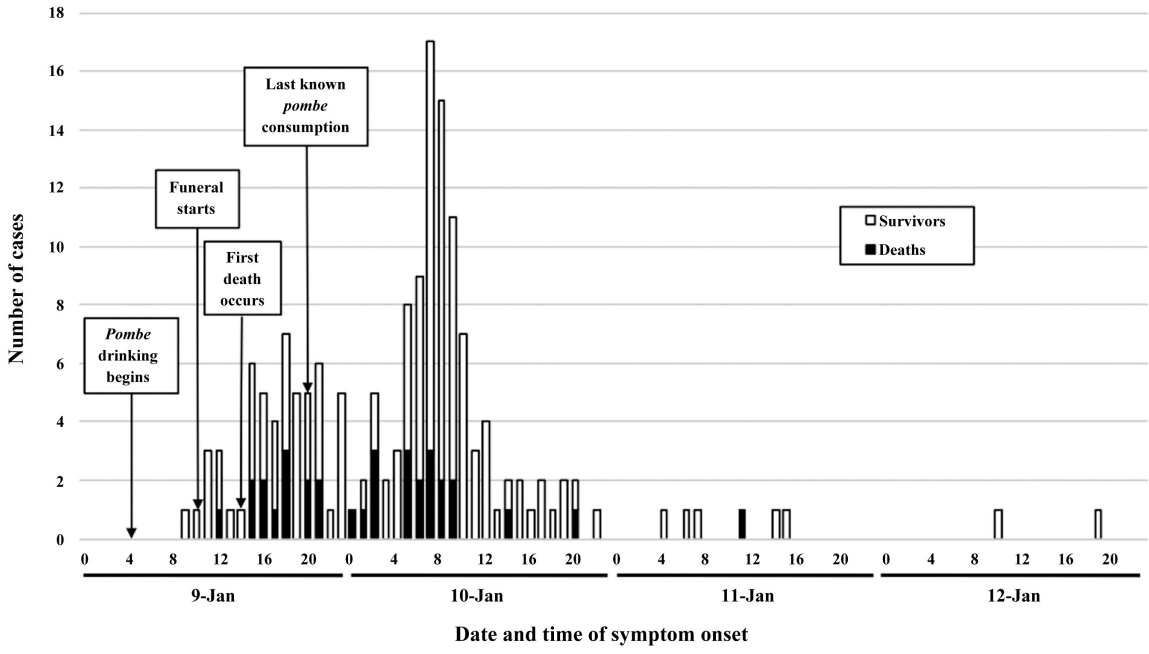

Figure 2. Time of illness onset among case patients by outcome, 9–12 January 2015. Time of illness onset information was available for 135 of 159 survivors (85%) and 33 of 76 who died (43%); times are shown as hours on a 24-hour clock.

Table 3. Food and *Pombe* Consumption of Noncase Patients and Case Patients by Disease Outcome

		Case Patients by Outcome			
	Noncase Patients (n = 42)	Recovered, Not Hospitalized (n = 107)	Recovered, Hospitalized (n = 52)	Death (n = 75)	*P* Value[a]
Food and drink, No. (%)	n = 41	n = 107	n = 52	n = 46	
Pombe[b]	35 (83)	106 (99)	51 (98)	75 (100)	.50
Xima	35 (85)	89 (83)	45 (87)	17 (37)	<.0001
Water	14 (34)	31 (29)	28 (54)	0 (0)	<.0001
Vegetables	10 (24)	29 (27)	15 (29)	8 (17)	.35
Beans	9 (22)	27 (25)	10 (19)	3 (7)	.02
Fish	12 (29)	28 (26)	13 (25)	5 (11)	.055
Bread	8 (20)	9 (8)	3 (6)	0 (0)	.12
Time of *pombe* consumption, No. (%)	n = 35	n = 81	n = 36	n = 26	
Morning	21 (75)	52 (64)	17 (47)	11 (42)	.03
Afternoon	7 (25)	29 (36)	19 (53)	15 (58)	
Estimated quantity of *pombe* consumed, median (range), mL[c]	130 (65–1800)	235 (50–1800)	225 (50–1700)	450 (130–2550)	.003
Estimated *pombe* dose, median (range), mL/kg)[d]	2.7 (1–24)	4.5 (1–33)	4.2 (1–31)	7.9 (2–45)	.005

[a] *P* values represent comparison between case outcome groups (not hospitalized, hospitalized, death).

[b] Data on *pombe* consumption were confirmed for all patients who died (all 75 consumed *pombe*) and noncase patients (35 of 42 persons who were not case patients consumed *pombe*).

[c] The estimated quantity of *pombe* consumed was provided by close relatives of case patients who died.

[d] For case patients who died, the weight used to calculate *pombe* dosage was imputed using the mean weight of all adult case patients of the same sex.

flour used to prepare the *pombe* was donated by a neighbor, who reported that the flour had been unintentionally soaked during recent floods. The corn flour was not used to prepare other foods or beverages other than the *pombe*.

The team considered many potential causes of the outbreak, including infectious agents, such as botulism, and chemical agents, particularly pesticides, cyanogenic glycosides, and heavy metals (eg, arsenic, cadmium, lead, and thallium). A literature review of indigenous toxic plants revealed none potent enough to cause a mass poisoning; interviews with traditional healers in Chitima supported the literature. We identified rodenticides, insecticides, fungicides, and solvents for sale at multiple locations in Chitima and in the provincial capital, Tete City (located 140 km from Chitima), none of which are associated with a syndrome consistent with the outbreak (Supplementary Table S3).

Laboratory Investigation

Pathologic examination of 2 deceased patients revealed livor mortis, bright red blood and organs, pulmonary edema with visceral congestion, and brain edema with resulting sulcal effacement and bilateral herniation of the cerebellar tonsils in both. Microscopic examination showed macro and microvesicular steatosis, periportal fibrosis, and periportal eosinophilic infiltration (Supplementary Figure S1). Pathologists also reported acute tubular necrosis and focal areas of necrosis in the gastric mucosa and pancreas.

Analyses of the flour and *pombe* samples at the US FDA Forensic Chemistry Center confirmed the presence of bongkrekic acid (BA) in the suspect *pombe* (range, 18–35 μg/mL) and corn flour (21 ng/g), as well as 2 other toxins, isobongkrekic acid (iBA) and toxoflavin (Supplementary Table S4). All 3 toxins are preformed mitochondrial toxins produced by *Burkholderia gladioli* pathovar (pv) *cocovenenans*. The control *pombe* was negative for all 3 toxins. A *Burkholderia* species was isolated from the corn flour; phylogenetic analysis suggested *B. gladioli* as the closest relative. In addition, the fungus *Rhizopus oryzae* was isolated from the corn flour. The isolated *Burkholderia* strain produced BA in vitro when coplated with the isolated *R. oryzae*, identifying the bacteria as the *B. gladioli* pv *cocovenenans*. A full list of laboratory results can be found in Supplementary Table S4.

DISCUSSION

This mass poisoning event was the first report of BA poisoning occurring outside Asia; our investigation yielded the most detailed epidemiologic and clinical description of a BA outbreak to date. We linked a mass poisoning event in rural Mozambique to consumption of a traditional beverage, *pombe*. We conclude that this outbreak was due to poisoning with BA following accidental bacterial contamination of corn flour used to make the *pombe*. Several lines of evidence support this conclusion. BA was detected in the consumed *pombe* at toxic levels and was not detected in the control *pombe* (Supplementary Table S4). *Burkholderia gladioli* was isolated from the corn flour used to prepare the *pombe*. Moreover, we found a dose-response relationship between the amount of *pombe* consumed and illness severity. Finally, the reported clinical manifestations were the same as those seen in previously described cases of

BA poisoning, including abdominal pain, diarrhea, vomiting, weakness, and palpitations [3].

Burkholderia gladioli pv *cocovenenans*, a gram-negative bacteria, was first isolated in 1932 from fermented coconut-based tempeh (*tempe bongkrek)* that caused a mass poisoning event in Indonesia [4]. The bacteria produces 3 toxins: BA, iBA, and toxoflavin [4, 5]. Of these, BA is the most potent toxin; doses as small as 1.0–1.5 mg have been reported to cause death [6]. Warm temperatures (22°C–30°C), neutral pH, and the presence of fungal organisms (notably *Rhizopus oligosporus* and *R. oryzae*, used in fermentation), support both bacterial growth and BA formation. Fatty acids, such as those in coconut and corn, are also required for toxin formation. BA and iBA are potent mitochondrial toxins. While the clinical manifestations and pathologic findings we observed were similar to those caused by other mitochondrial toxins (eg, cyanide), BA and iBA do not interfere directly with the electron transport chain. Rather, they inhibit the adenine nucleotide translocator within the inner mitochondrial membrane, thereby blocking the ingress and phosphorylation of adenosine diphosphate to adenosine triphosphate and ultimately halting aerobic respiration [7].

Reports of BA poisoning were previously limited to 2 scenarios: after consumption of coconut-based tempeh in Java, Indonesia, and after consumption of fermented flour or mushrooms in China [3, 8, 9, 10]. Traditionally prepared coconut tempeh was banned in Indonesia because of the risk of poisoning, but occasional outbreaks associated with illegally produced tempeh continue to occur [8, 10].

The outbreak in Mozambique is similar to previous BA poisonings in terms of route of exposure, mortality rate, and clinical manifestations [4, 8, 9, 11–13]. We observed a case fatality rate of 32% in the Chitima outbreak. In previous outbreaks, case fatality rates of up to 100% have been reported, but most range from 30% to 40% [11]. The progressive neurologic deterioration that we observed in some patients most likely resulted from increased intracranial pressure from cerebral edema, which is also a manifestation of cyanide poisoning [14]. Our investigation is the first to document health effects from BA after the acute poisoning period, with more than three-quarters of interviewed survivors continuing to experience sequelae 1 month after exposure.

Two individuals met the case definition but did not consume *pombe.* Our belief is that their diseases were due to some other cause, because other diseases endemic in the area can present with similar signs and symptoms [15, 16]. One patient presented with a self-limited headache. The other patient probably had acute gastroenteritis, a common condition in patients seen at primary healthcare facilities in Mozambique.

It is unknown how the corn flour became contaminated with *B. gladioli* pv *cocovenenans*. Traditional processing of corn flour in Mozambique and other African countries often involves drying the flour on mats placed on the bare ground. We hypothesize that the floodwater that soaked the suspect corn flour was contaminated with *B. gladioli* pv *cocovenenans* from the soil. Although *pombe* is boiled several times during its production, BA is a heat-stable toxin that would not be degraded by high temperatures [6]. Detailed steps of the *pombe* preparation process are provided in the Supplementary Appendix. *Pombe* production was banned in Chitima for a 3-month period after the outbreak, but it continued regardless, because it is an important source of income in this community.

As noted in Supplementary Table S4, aflatoxin B1 was detected in the suspect *pombe* (range, 5.9–20 ng/g) but not in the control *pombe*. Aflatoxins are a frequent contaminant of corn in Tete Province [17]; however, the levels of aflatoxin B1 in the *pombe* would not be expected to cause acute aflatoxin poisoning [18].

The current investigation had several limitations. The sudden presentation of numerous patients with severe and unexplained illness overwhelmed local resources, complicating timely and appropriate data collection; therefore, clinical records were limited or unavailable for several patients. Similarly, owing to the limited number of pathologists in Mozambique, only 2 autopsies were performed. Patients and family member proxies were asked to remember how much *pombe* was consumed, often a week after the exposure, potentially introducing recall bias. Therefore, the observed dose-response trend between increased illness severity and increased *pombe* consumption should be interpreted with caution.

Another limitation was that the follow-up investigation was not based on a random sample; the investigation was limited to the 2 most affected neighborhoods, and community leaders were asked to identify survivors 1 month after the outbreak, possibly leading to selective reporting of those who were experiencing continued symptoms or had been notably sick. In addition, because *pombe* production had been banned in Chitima, the control *pombe* was collected in another village. Although not an ideal control, the *pombe* from the neighboring village allowed laboratory analysts to identify compounds not present in both samples and subsequently identify the toxins in the suspect *pombe*. The identification of BA in the *pombe* would ideally be corroborated by finding BA or a metabolite in patient samples; however, no clinical assay exists for BA.

Although BA poisoning has never been reported outside of Asia, it is possible that smaller outbreaks, with less severe disease presentation and fewer fatalities, have occurred in the past and gone unnoticed. Though *B. gladioli* is found globally in a variety of ecologic niches, including soil [19, 20], little is known about the distribution of the *cocovenenans* pathovar or its interactions with specific endemic strains of *Rhizopus* spp. The global presence of this microorganism could have significant public health implications, especially in sub-Saharan Africa, where consumption of traditional, cereal-based fermented beverages similar to *pombe* is widespread [21–23]. Sanitary conditions during

production, storage, and consumption are precarious; contamination of traditional beverages by microbial pathogens [24], aflatoxins [24], and heavy metals [24] has been documented. Strengthening food safety standards and providing guidance to local brewers are prudent steps to decrease the risk of contamination with *B. gladioli* and other contaminants of concern.

This investigation contributes to the limited body of knowledge on the occurrence and epidemiology of BA poisoning by describing the first reported outbreak from Africa. We believe it is possible that unrecognized outbreaks caused by the toxin have occurred elsewhere outside of Asia. Additional research is needed to understand the geographic distribution of *B. cocovenenans* in order to avoid similar events in the future, particularly in regions where homemade alcoholic beverages are consumed.

Supplementary Data

Supplementary materials are available at *Clinical Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

Notes

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Disclaimer. The findings and conclusions in this report do not necessarily represent the official views of the CDC, the US Food and Drug Administration (FDA), or the Association of Schools and Programs of Public Health. The funders of the investigation had no role in study design, data collection, data analysis, data interpretation, or the writing of the report. The corresponding author had full access to all the data in the study and final responsibility for the decision to submit for publication.

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