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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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Abstract

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

Methods—An investigation was conducted to identify cases and determine the cause of the outbreak. A case was defined as any resident of Chitima who developed any new or unexplained neurologic, gastrointestinal, or cardiovascular symptom from January 9 at 6:00 a.m. through

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January 12. We conducted medical record reviews; healthcare worker and community surveys; anthropological and toxicological investigations of local medicinal plants and commercial pesticides; and laboratory testing of the suspect and control *pombe*.

Results—We identified 234 cases; 75 (32%) died and 159 recovered. Overall, 61% of cases were female (n=142), and ages ranged from 1–87 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 in 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 of Asia, our investigation suggests that BA might be an unrecognized cause of toxic outbreaks globally.

Keywords

Bongkrekic acid; Burkholderia gladioli; foodborne; outbreak; poisoning

Background

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

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 health care 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 (MMOH), which dispatched an investigation team to Chitima on January 12.

Methods

Epidemiologic Investigation

A multidisciplinary team comprised of representatives from the MMOH, the Tete Provincial Health Department, the US Centers for Disease Control and Prevention (CDC), and the World Health Organization (WHO), investigated the outbreak. A case was defined as any Chitima resident who developed any new or unexplained neurologic, gastrointestinal, or

cardiovascular symptom (Table 1) from January 9 at 6:00 a.m. through January 12 at 11:59 p.m.

We identified potential cases by i) reviewing the medical records of all patients presenting during January 9–12, 2015 and interviewing clinicians at the Chitima Health Center and Songo Rural Hospital ii) interviewing hospitalized patients, and iii) soliciting lists of persons who had become sick or died from local community leaders. Deaths were confirmed with health care providers and family members of the deceased.

We interviewed affected individuals during January 13–19, 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 prior to symptom onset. Family members were interviewed as proxies for the deceased. The clinicians who cared for the initial patients reported that many affected individuals had a recent history of drinking *pombe*; therefore, interviewers provided a series of commonly used cups of known volume to assist individuals in estimating the quantity of *pombe* consumed. Ultimately, some interviewees did not meet the case definition; these individuals were chosen as a convenience sample of non-cases to compare to cases regarding potential exposures.

To assess any continuing sequelae four weeks after the poisoning event, during February 5–7, 2015, the team interviewed and examined 17 survivors identified by community leaders. During this visit, the team conducted a toxicological field investigation by visiting the Chitima District Office of Economic Activities and vendors at informal markets to identify locally available toxic chemicals, conducting a literature review to identify endemic toxic plants, and consulting local traditional healers to discuss local home remedies and poisons. We 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 two 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 January 10th; 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 U.S. Food and Drug Administration (USFDA) Forensic Chemistry Center laboratory expanded testing of the *pombe* and corn flour samples using a battery of forensic screening tests. These tests included non-targeted and quantitative liquid chromatography-mass spectrometry (LC/MS) and microbiological testing [2].

Statistical Analysis

Using Epi Info (version 7.1.3.10, CDC, 2012) and SAS 9.3 (SAS Institute, Cary, NC), we analyzed potential associations between demographic factors and food and beverage consumption with illness and death using the chi-square test and Fisher's exact test for categorical variables. Severity of illness was categorized as recovered, not hospitalized; recovered, hospitalized; and death. For continuous variables, we performed t-tests for normally distributed and Wilcoxon rank-sum tests for non-normally distributed variables. We used the Kruskal-Wallis test to evaluate the dose-response relationship between the amount of *pombe* consumed and clinical outcomes. Alpha ≤0.05 was considered statistically significant.

Role of the Funding Source

The funder of the investigation had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Epidemiologic Investigation

We identified 234 people who met the case definition. Overall, 61% of the people identified as cases were female (n=142) (Table 2). Age ranged from 1–87 years, with a median age of 30 years. Increasing age was associated with death (p=0.0029). Affected individuals resided in all ten neighborhoods of Chitima; however, the majority (n=111) lived in the Cawira B neighborhood, where the *pombe* was prepared and sold. Of the 234 cases, 103 were hospitalized and 75 died (case-fatality rate: 32.1%). Fifty-one patients (68.0%) who died had been hospitalized; the remaining 24 (32.0%) who died did not seek medical attention, likely because they were too ill to walk to a health facility. The earliest reported symptom onset was January 9 at 4:00 p.m. The first recorded death was overnight on January 9, and the last recorded death occurred on January 16 (Figure 2).

Data regarding the time of *pombe* consumption and time of symptom onset were available for 120 (51.3%) patients. The median time to symptom onset was 16 hours after *pombe* consumption (range: 0–148). Clinical data were available for 205 (87.6%) individuals. 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 hyper-salivation, diaphoresis, or miosis were noted. Patients who died developed progressive confusion, loss of consciousness, abnormal posturing, and death. Some gravely ill patients had rigidity on 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 Table S1 in the Supplementary Appendix. Four weeks later, 13/17 (76%) interviewed survivors reported ongoing symptoms, including neurologic signs and symptoms (e.g., 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 cases (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 were classified as cases. Compared with the 42 people who were not cases, those who were cases had greatly increased odds of consuming *pombe* (OR 23.2; 95% CI = 4.6–116.2). Only two individuals meeting the case definition denied consuming *pombe*. Neither was gravely ill and both had only one qualifying symptom. Those who died drank more *pombe* per body weight compared to those who survived (median mL/kg: 7.9 versus 4.5, p=0.0013) (Table 3). Those who died had a shorter time interval between drinking *pombe* and disease onset compared to survivors (12 hours versus 17 hours, p=0.0036).

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 (Table S2 in the Supplementary Appendix).

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 a.m. to be sold at the funeral ceremony; she was not seen again before being found dead in her home at 2:00 a.m. the following morning. The corn 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. 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 (Table S3 in the Supplementary Appendix).

Laboratory Investigation

Post-mortem examination of two patients revealed livor mortis, bright red blood and organs, pulmonary edema with visceral congestion, and brain edema with resulting sulcal effacement and bilateral tonsillar herniation in both. Microscopic examination showed macro and microvesicular steatosis, periportal fibrosis, and periportal eosinophilic

infiltration (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 USFDA Forensic Chemistry Center detected bongkrekic acid (BA) in the suspect *pombe* (range: 18–35 μg/mL) and corn flour (21 ng/g) as well as two other toxins, isobongkrekic acid (iBA) and toxoflavin (Table S4). All three toxins are preformed mitochondrial toxins produced by *Burkholderia gladioli* pathovar (pv.) *cocovenenans*. The control *pombe* was negative for all three toxins. A *Burkholderia sp.* 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 co-plated with the isolated *R. oryzae*, identifying the bacteria as *Burkholderia gladioli* pv. *cocovenenans*. A full list of laboratory results can be found in Table S4 in the Supplementary Appendix.

Discussion

This mass-poisoning event was the first report of BA poisoning occurring outside of Asia; our investigation yielded the most detailed epidemiological 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* (Table S4). *B. 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 [9].

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 [3]. The bacteria produces three toxins: BA, iBA, and toxoflavin [3, 4]. Of these, BA is the most potent toxin; doses as small as 1.0–1.5 mg have been reported to cause death [5]. Warm temperatures (22–30°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 features we observed were similar to those caused by other mitochondrial toxins (e.g., 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 (ADP) to adenosine triphosphate (ATP) and ultimately halting aerobic respiration [6].

Reports of BA poisoning were previously limited to two scenarios: after consumption of coconut-based tempeh in Java, Indonesia, and after consumption of fermented flour or mushrooms in China [7–10]. 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 [7, 10].

The outbreak in Mozambique is similar to previous BA poisonings in terms of route of exposure, mortality rate, and clinical manifestations [3, 7, 8, 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 30–40% [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 over three quarters of interviewed survivors continuing to experience sequelae one month post-exposure.

Two individuals met the case definition but did not consume *pombe*. Our belief is that their diseases were due to some other etiology, as 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 likely had acute gastroenteritis, a common condition in patients seen at primary health care facilities in Mozambique.

It is unknown how the corn flour became contaminated. 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. Though *pombe* is boiled several times during production, BA is a heat-stable toxin that is not degraded by high temperatures [5]. Detailed steps of the *pombe* preparation process are in the Supplementary Appendix. *Pombe* production was banned in Chitima for a three-month period following the outbreak; regardless, *pombe* production continued in Chitima throughout this period, since it is an important source of income in this community.

As noted in Table S4 in the Supplementary Appendix, 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 aflatoxin B1 levels in the *pombe* would not be expected to cause acute aflatoxicosis [18].

This 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, due to the limited number of pathologists in Mozambique, only two 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. The follow-up investigation was not based on a random sample; the investigation was limited to the two most affected neighborhoods, and community leaders were asked to identify survivors one month after the outbreak, possibly leading to selective reporting of those who were experiencing continued symptoms or had been notably sick. In addition, since *pombe*

production had been banned in Chitima, the control *pombe* was collected in another village. While 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.

While 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 (storing grains above the ground in a dry, clean environment, avoiding the addition of uncooked corn at the end of cooking, and using clean equipment for *pombe* preparation) 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 Material

Refer to Web version on PubMed Central for supplementary material.

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Key points

We investigated a mass poisoning event that killed 75 people in Mozambique. We detected bongkrekic acid in a communal beverage and corn flour used to make it. We also isolated the causative bacteria, Burkholderia gladioli pathovar cocovenenans, from the flour.

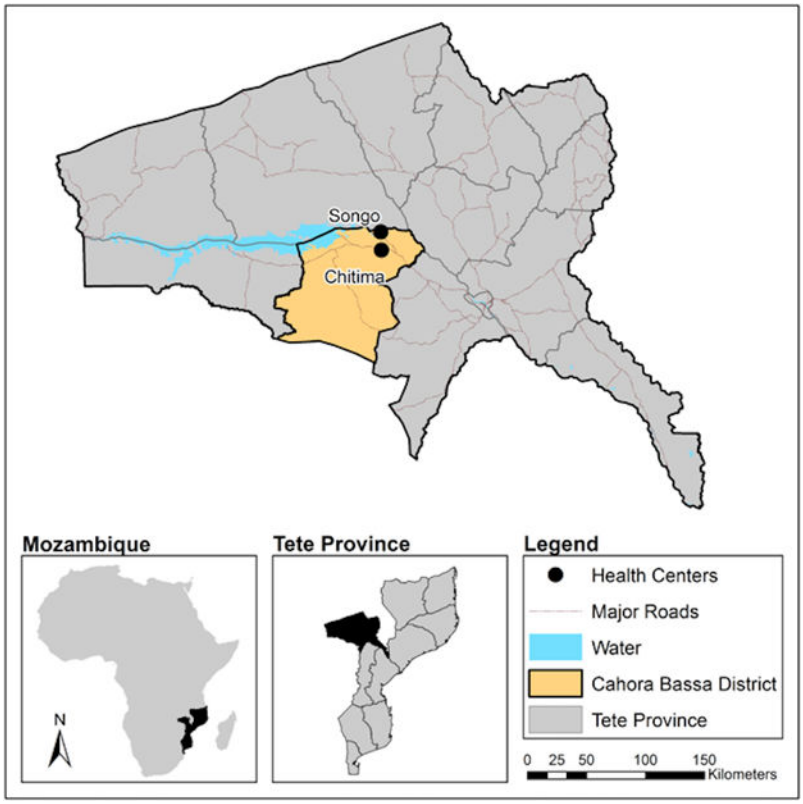

Figure 1.
Location of outbreak, Cahora Bassa District, Tete Province, Mozambique

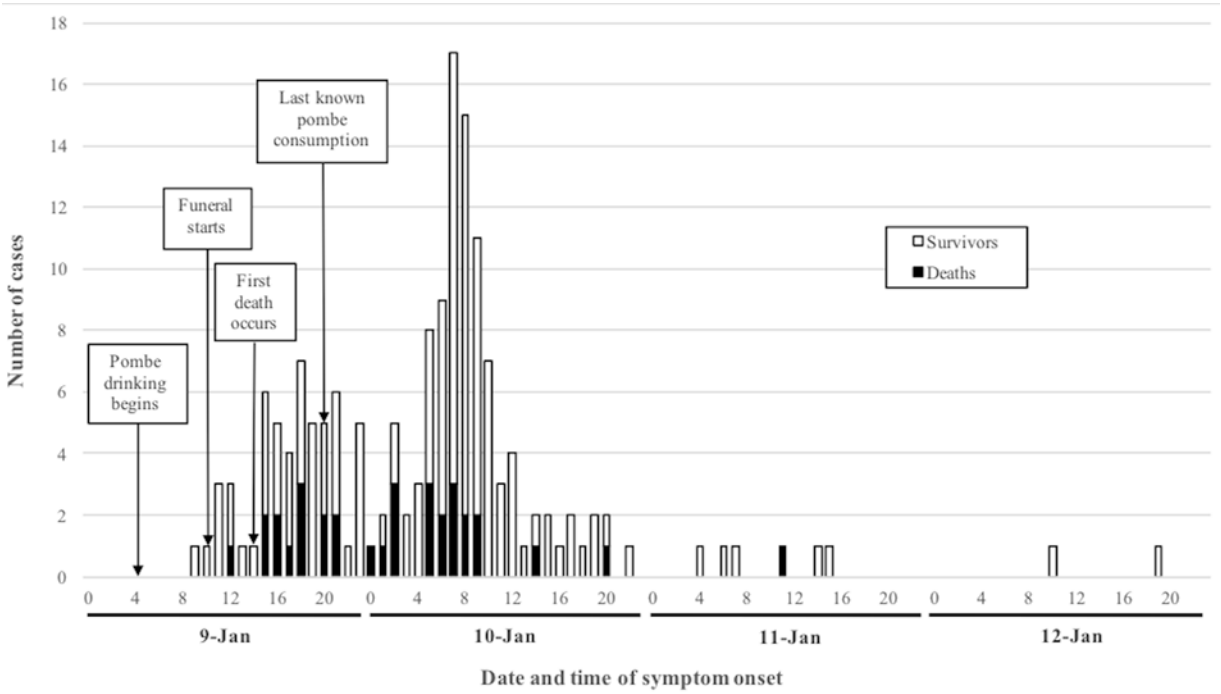

Figure 2. Time of illness onset among case-patients by outcome, January 9–12, 2015

Time of illness onset information was available for 135/159 (85%) survivors and 33/76 (43%) deaths.

Table 1

Case definition - Any resident of Chitima who developed any new or unexplained symptom from the following list from January 9, 2015 at 6:00 a.m. through January 12, 2015 at 11:59 p.m.:

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

Table 2

Demographic characteristics and clinical manifestations of 234 Chitima residents with disease onset during January 9–12, 2015

	Recovered, Not Hospitalized	Recovered, Hospitalized	Death	All Cases
Total	**107**	**52**	**75**	**234**
Sex (N [%])				
Male	38 (36%)	21 (40%)	33 (44%)	92 (39%)
Female	69 (64%)	31 (60%)	42 (56%)	142 (61%)
Age (years)[a]				
Range	1–87	6–80	3–78	1–87
Median	29	32	39	30
Unknown	4	4	19	27
Signs and Symptoms (N [%])	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[b]	5 (5%)	1 (2%)	13 (28%)	19 (9%)
Psychomotor agitation[b]	2 (2%)	0 (0%)	14 (30%)	16 (8%)

[a]Differences in mean age among the three outcome groups were statistically significant (p=0.0125) by Kruskal-Wallis test

[b]These signs were clinician-reported and were not included in the case questionnaire

Table 3

Food and *pombe* consumption of non-cases and cases by presence and severity of the disease

		Cases			
	Non-cases	**Recovered, Not Hospitalized**	**Recovered, Hospitalized**	**Fatal**	**P value[a]**
Totals	**42**	**107**	**52**	**75**	–
Food & drink N (%)	**n = 41**	**n = 107**	**n = 52**	**n = 46**	–
Pombe[b]	35 (83%)	106 (99%)	51 (98%)	75 (100%)	0.4976
Xima	35 (85%)	89 (83%)	45 (87%)	17 (37%)	<0.0001
Water	14 (34%)	31 (29%)	28 (54%)	0 (0%)	<0.0001
Vegetables	10 (24%)	29 (27%)	15 (29%)	8 (17%)	0.3545
Beans	9 (22%)	27 (25%)	10 (19%)	3 (7%)	0.0209
Fish	12 (29%)	28 (26%)	13 (25%)	5 (11%)	0.0552
Bread	8 (20%)	9 (8%)	3 (6%)	0 (0%)	0.1168
Time of pombe consumption	**n = 35**	**n = 81**	**n = 36**	**n = 26**	–
Morning	21 (75%)	52 (64%)	17 (47%)	11 (42%)	0.0267
Afternoon	7 (25%)	29 (36%)	19 (53%)	15 (58%)	
Estimated quantity of pombe consumed (mL)[c]					
Range	65–1800	50–1800	50–1700	130–2550	0.0031
Median	130	235	225	450	
Estimated pombe dose (mL/kg)[d]					
Range	1–24	1–33	1–31	2–45	0.0054
Median	2.7	4.5	4.2	7.9	

[a] P values are for comparison between case outcome groups (not hospitalized, hospitalized, fatal)

[b] Data on *pombe* consumption was confirmed for all deaths (75/75 fatal cases consumed *pombe*) and non-cases (35/42 people who were not cases consumed *pombe*)

[c] Estimated quantity of *pombe* consumed was provided by close relatives of fatal cases

[d] For fatal cases, the weight used to calculate *pombe* dosage was imputed using the mean weight of all adult cases of the same gender