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Outbreak of type A foodborne botulism at a boarding school, Uganda, 2008

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SUMMARY

Botulism has rarely been reported in Africa. In October 2008, botulism was reported in three Ugandan boarding-school students. All were hospitalized and one died. A cohort study was performed to assess food exposures among students, and clinical specimens and available food samples were tested for botulinum toxin. Three case-patients were identified; a homemade, oil-based condiment was eaten by all three. In the cohort study, no foods were significantly associated with illness. Botulinum toxin type A was confirmed in clinical samples. This is the first confirmed outbreak of foodborne botulism in Uganda. A homemade, oil-based condiment was the probable source. Consumption of homemade oil-based condiments is widespread in Ugandan schools, putting children at risk. Clinicians and public health authorities in Uganda should consider botulism when clusters of acute flaccid paralysis are seen. Additionally, schools should be warned of the hazard of homemade oil-based condiments, and take steps to prevent their use.

Key words: Botulism, food poisoning, outbreaks.

INTRODUCTION

Foodborne botulism is a rare, paralytic disease caused by ingestion of toxin made by neurotoxigenic *Clostridium* spp., which are spore-forming bacteria found in soil and aquatic sediments. The toxin binds irreversibly to the neuromuscular junction, causing cranial nerve palsies and descending flaccid paralysis that can lead to respiratory failure and death. Foodborne botulism is a public health emergency, as a single contaminated food can cause illness in many people.

In October 2008, three cases of botulism were suspected in residents of a dormitory at a boarding school in Kampala, Uganda. To our knowledge, botulism has never been reported from Uganda, and foodborne botulism has rarely been reported from Africa [1–5]; documented outbreaks have involved fermented uneviscerated fish [1], soured milk [2], termites sealed in a bag [3], tinned fish [4] and sausages [5]. In response to the suspected outbreak, the Uganda Ministry of

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Health (UMOH), the World Health Organization (WHO), and the Centers for Disease Control and Prevention (CDC) conducted an emergency public health investigation.

METHODS

Case definition and case-finding

A case was defined as illness in an individual residing or working at the school whose clinical presentation included at least one cranial nerve palsy and bilateral extremity weakness/paralysis, without fever or altered mental status, as well as botulinum toxin or botulinum toxin-producing *Clostridium* spp. in a clinical specimen, or having consumed the same implicated food as a laboratory-confirmed case, during October 2008. Suspect cases were examined and interviewed, and additional case-finding was performed at the school through interviews with the school nurse and review of school clinic records. Additionally, neurologists and intensive-care physicians at Mulago Hospital, the national referral hospital for Uganda, were asked about other patients exhibiting signs and symptoms consistent with botulism. Queries were made within the region where the school was located, and nationally for increased reports of acute flaccid paralysis (AFP) during October 2008.

Treatment

Botulism antitoxin, the only specific treatment for botulism, is not available in Uganda. Botulism antitoxin can halt the progression of symptoms, if administered early in the course of disease. CDC provided botulism antitoxins A, B, and E for treatment of suspect cases. Antitoxin was administered by Ugandan clinicians according to the manufacturers' protocols.

Environmental assessment, hypothesis generation and cohort study

The boarding school area, including the dormitory where case-patients resided and areas of food preparation and food service, was surveyed. In-person interviews with case-patients, their parents, and other dormitory residents were performed. Seven-day food histories were collected from surviving case-patients. Initial interviews were used to build a cohort analysis instrument to further evaluate the association between illness and food exposures. The cohort was defined as all residents of the dormitory where the case-patients lived. The exposure period was 7 days before the first case onset date.

Laboratory evaluation

Clinical samples (serum or stool) from suspect case-patients and suspect foods were preliminarily tested at the Uganda Virus Research Institute in Entebbe, Uganda, and definitively tested at CDC, Atlanta. Mouse bioassay and antitoxin neutralization tests for detection of botulinum toxin, and bacterial culture for isolation of neurotoxigenic *Clostridium* spp. were performed according to established protocols [6].

Statistical methods

Data were collected using a Microsoft Access database (version 2003, Microsoft Corporation, USA). Analyses were performed using SAS version 9.1 (SAS Institute Inc., USA). Univariate analysis was performed, including the calculation of risk ratios and 95% exact confidence intervals, where appropriate.

RESULTS

Initial response

An outbreak was suspected when clinicians at the Mulago Hospital in Kampala reported two suspect cases to the UMOH, who initiated an outbreak investigation in collaboration with WHO. Shortly thereafter, CDC headquarters in Atlanta, GA were notified on 30 October 2008 by CDC offices in Uganda of three possible suspect botulism cases. An invitation was extended to the CDC to join the ongoing UMOH and WHO investigation; the CDC team arrived with antitoxin in Uganda on 2 November 2008. The investigation continued in the weeks following, including an environmental investigation, extensive interviews and ultimately a cohort study. Interviews for the cohort study were completed by 15 November 2008.

Case-patients and treatment

All case-patients were females aged 17–18 years who lived in the same dormitory at the boarding school and had illness onsets between 19 and 22 October 2008. One case-patient died on 24 October 2008, 4 days after illness onset, at a hospital without

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mechanical ventilators. The other two case-patients were admitted to the national referral hospital on 23 and 29 October 2008, where one required mechanical ventilatory support; it is likely that the availability of intensive supportive care, including mechanical ventilation, was a key factor in the survival of those two patients. Both surviving patients received botulism antitoxins A, B, and E without adverse events, and were later discharged home with improved neurological function. Attempts were made to identify other possible cases at the school through interviews with the school nurse and review of her urgent care logs, and more widely through queries to referral hospitals, within the region where the school was located, and nationally for increased reports of AFP during October 2008. No other possible cases were identified.

Environmental assessment and hypothesis generation

The boarding school housed 1200 students from September to December 2008. School dormitories usually hold between 18 and 24 students of the same gender, and 23 females (including the case-patients) lived in the dormitory where the case-patients lived. Meals were prepared and served to all students from a central kitchen and consumed within dormitories. There were no easily accessible sources of outside food during the school term; however, students commonly brought commercially produced and homemade food in their suitcases at the start of the term, and shared food regularly within the dormitory. Various seasonings (called 'appetizers' by the students) were commonly used by students to flavour school food. These seasonings used throughout the term, were freely shared among students, particularly within dormitories, and included locally made peanut butter and a variety of homemade oil-based condiments flavoured with vegetables. Quantities varied, from smaller jars of solids (<500 g) up to 3-litre 'jerry cans' of liquids. One such oil-based condiment (condiment A) had been prepared by a case-patient before the start of the school term and stored at room temperature since the beginning of the term. Condiment A was a semi-solid mixture of commercial vegetable fat, fried onions, ginger, peppers, and spices; the fat was heated until it began 'smoking', the washed and chopped vegetables were added, and then the resulting mixture was poured into a plastic container and allowed to cool. During the term, 1–2 scoopfuls of condiment A were mixed into food provided by the school. Multiple individuals, including both surviving case-patients, reported seeking out onion or peppers mixed into condiments for their flavour. Although dormitory residents reported consuming other homemade foods, we deduced, based on interviews with surviving case-patients, that the only homemade food probably eaten by all case-patients was condiment A. Some dormitory residents reported that condiment A 'did not taste [or smell] good'. At the time of the investigation, condiment A was no longer available for inspection. From reports made by students and case-patients, multiple condiments were running out or ran out during the period just prior to illness onsets. Of note, mould was observed on the surface of oil-based condiments being consumed at the school at the time of the investigation.

Cohort study

Twenty (87%) of 23 dormitory-mates were interviewed; among non-responders, one was the deceased student, and the other two were students who were absent due to illnesses not clinically consistent with botulism. The median age was 18 years. No foods were significantly associated with illness ($P \geq 0{\cdot}10$), although 15/20 (75%) of dormitory-mates consumed at least one oil-based condiment during the week prior to the case-patients' illness onsets. When asked about their food-eating preferences, 17/20 (85%) students reported adding oil-based condiments to their meals. Evaluation of dose through number of meals exposed and amount of condiment A added per meal did not reveal a dose–response relationship between illness and condiment consumed. Although the case-patient who made condiment A had previously reported consuming condiment A during the exposure period, in the cohort study she denied consuming condiment A and was classified as unexposed to condiment A in the analysis. However, six dormitory-mates, one ill and five well, reported consuming condiment A during the exposure period.

Laboratory evaluation

Botulinum toxin was detected in the sera of the two surviving case-patients, but serum quantities were insufficient to determine toxin type. Enrichment cultures of stool from one case-patient yielded *Clostridium botulinum* toxin type A. Specimens could not be obtained from the deceased case-patient.

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Condiment A had been consumed and its container discarded before samples could be obtained. Locally made peanut butter, margarine, four types of processed juice and residue in a container from another type of oil-based condiment found in the dormitory room all tested negative.

DISCUSSION

We describe the first known outbreak of foodborne botulism in Uganda, which caused severe type A botulism, including one fatality, among three dormitory-mates at a boarding school. Although we were unable to confirm the source of toxin, the most likely source was a homemade, oil-based condiment available to residents of the dormitory room where the three cases occurred. Initial interviews indicated that all three case-patients consumed condiment A before their illness, although the case-patient who prepared condiment A later denied consuming it during her cohort study interview. It is unlikely that a school meal served to all 1200 students or a nationally distributed commercial product caused the outbreak, because illness was localized in residents of a single dormitory of the school. Future outbreaks at the school remain a threat because a large proportion of students regularly consume homemade oil-based condiments stored unrefrigerated.

Public health authorities should suspect botulism when clusters of AFP are reported in Uganda and other countries, even those where botulism has not been previously described. Prompt public health investigation and identification of contaminated foods are critical to prevent additional illnesses. Botulism has been infrequently reported from other regions in Africa, but not from foods similar to the suspected vehicle in this outbreak. Given the widespread consumption of homemade oil-based condiments in Uganda, we cannot explain why botulism has not been previously reported from Uganda.

Storage of homemade oil-based condiments in the dormitory for up to 2 months at room temperature in a sealed container may have promoted germination and production of botulinum toxin by *C. botulinum* spores, which may have been present in the original ingredients, or introduced during repeated use of the food. Obvious spoilage was observed in another flavoured condiment consumed at the school; although this does not support the presence of *C. botulinum* contamination specifically, it does indicate microbial contamination and inadequate storage conditions. Condiment A and other condiments in the dormitory contained peppers, onions, and other vegetables that are grown in soil and can harbour *C. botulinum* spores. Fats and oils can provide the anaerobic, low-acidity environment necessary for spore germination and toxin elaboration [7–10]; repeated outbreaks of garlic-in-oil preparations in the USA led the US Food & Drug Administration to enact more stringent guidelines for commercial oil manufacture to include both acidification and refrigeration as barriers to *C. botulinum* spore germination [10].

Our investigation has several limitations. We attempted to minimize recall bias with memory aids during interviews, and emphasized that no single individual would be punished for supplying a particular homemade food. However, there may still have been fear of reprisals from the school or fellow students, and we were unable to conclude whether discrepant historical information resulted from problems with recall, fear of reprisals, or other factors. Moreover, if condiment A caused the outbreak, we cannot fully explain why additional dormitory residents who consumed the oil-based condiment did not develop botulism symptoms, although micro-environments within the semi-solid condiment A could have allowed for differential exposures in dormitory residents. Further, absence of symptoms in some persons who consumed the implicated product is not uncommon in botulism outbreaks [11]. Last, our ability to confirm the source of toxin was limited, as some homemade foods, including condiment A, were not available for testing.

In summary, this is the first known foodborne botulism outbreak in Uganda. Botulism is a potentially fatal disease and represents both a medical and public health emergency. Even when antitoxin is promptly administered, neurological sequelae may take weeks to months to resolve [12], burdening patients, their families, and the healthcare system [13]. Although the source of ingested toxin in this outbreak was not confirmed, improperly stored homemade oil-based condiments were identified as a plausible and likely cause. We recommend that school officials enforce existing prohibitions on bringing homemade foods into the school, and particularly emphasize to students and families the risk of unrefrigerated oil-based condiments.

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DECLARATION OF INTEREST

None.

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