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A hospital acquired outbreak of *Bacillus cereus* gastroenteritis, Oman

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KEYWORDS

Bacillus cereus; Food poisoning; Nosocomial infection; Outbreak investigation; Oman

Abstract

Objectives: To investigate the course of a hospital acquired outbreak of *Bacillus cereus* gastroenteritis outbreak, and the interventions that were taken to prevent such an outbreak from occurring again.

Methods: On May 3–5 2008, 58 cases of gastroenteritis were reported among patients and their attendants in a referral hospital in Oman. All affected had eaten meals served by the hospital kitchen the previous day. An outbreak investigation team conducted active surveillance and interviewed people about symptoms and food consumed on the preceding day in the hospital. Food samples from the kitchen and faecal samples from the kitchen staff and those affected were cultured. An environmental audit of the kitchen was conducted.

Results: The majority of the 58 persons affected by the outbreak were adult females, predominantly attendants of patients. 90% had diarrhoea and 10% had vomiting, usually mild. All those affected were managed symptomatically except for two patient attendants who required intravenous rehydration. The meal exposure histories implicated at least one meal from the kitchen. Many violations of basic food hygiene standards were observed in the kitchen. Toxin producing *B. cereus* was isolated from faeces of 3/12 (25%) patients and 19/25 (76%) of food handlers, and 35/61 (57%) of food samples from the kitchen.

Conclusion: This is the first report of a nosocomial outbreak of foodborne *B. cereus* infection from this region. The importance of appropriate epidemiological and

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microbiological investigation and public relations management is emphasized, in addition to the need for continuing training of food handlers and rigorous enforcement of food hygiene regulations.

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Introduction

Bacillus cereus is widespread in nature and frequently isolated from soil and growing plants, but it is also well adapted for growth in the intestinal tract of insects and mammals. From these habitats it is easily spread to foods, where it may cause an emetic or a diarrhoeal type of foodassociated illness that is becoming increasingly important in the industrialized world [1]. B. cereus is a known cause of foodborne disease worldwide [2,3], although it is probably highly under-reported in official lists of foodborne disease causes. In the European Union, Bacillus species (including noncereus) were reported to be responsible for 1.4% of foodborne outbreaks in 2005. Between 1993 and 1998 in the Netherlands, B. cereus accounted for 12% of foodborne disease outbreaks in which a causative agent was identified [4]. The incubation period for B. cereus induced gastroenteritis is usually over 6h, normally in the range of 8-16h, and on average 12 h, but in rare cases longer incubation times have been observed. The duration of the disease is normally 12-24h but cases lasting several days have been reported [5].

On 4 May 2008, several people attended the adult emergency department of the hospital with symptoms of gastroenteritis, and patients and their relatives on several wards were reported to the Infection Prevention and Control Team (IPCT) to have similar symptoms. It seemed likely that an outbreak of food poisoning had occurred and a taskforce was immediately convened by the hospital management to investigate this and take appropriate action.

The outbreak, which affected at least 58 people, lasted 24h and was determined to be caused by food in the hospital kitchen being contaminated by *B. cereus*. This is the first reported nosocomial outbreak of food poisoning in Oman, and received widespread adverse publicity in the local press. This report outlines the investigation and course of the outbreak, and the interventions that were taken to prevent such an outbreak from occurring again.

Methods

Background

The Royal Hospital in Muscat has a capacity of 625 beds and is the main tertiary care hospital in Oman. On 4 May 2008 at 07:00 h the duty nursing officer was informed by different ward nurses that some of the patients' attendants in the hospital wards were suffering from symptoms consistent with gastroenteritis. Those affected were asked to attend the Emergency Department and the unusual number of patients attending suggested a point-source outbreak. All those whose illness was notified had eaten meals provided by the hospital kitchen, which is run by a local catering company. The catering company has a permanent staff of 25 employees and prepares around 3000 meals a day. The caterer's food handlers had taken a course on safe food preparation and the company is ISO 9001 certified and had been audited by an external team 2 months before the outbreak.

Epidemiological investigation

An outbreak investigation task force was immediately established, including nurse and the physician in charge of IPCT, representatives from microbiology, emergency room, nursing and hospital management. An epidemiological study was designed to investigate the outbreak, and active surveillance of patients, their attending relatives and staff was conducted throughout the hospital.

Because the common exposure of the persons who became ill seemed obvious, the prospective epidemiological investigation focused on the hospital kitchens. The caterer provided a list of food served at the hospital on the day before the onset of symptoms. An epidemiological case definition of an outbreak case was developed after analyzing the initial data available from the first patients who attended the Emergency Department. An outbreak case was defined as ''an inpatient or any person who was an attendant of a patient admitted to the Royal Hospital, presenting with one or more of the following symptoms: diarrhoea, abdominal pain or vomiting''.

The nurse in charge of each ward asked all patients and attendants on their ward about symptoms of gastroenteritis. This was repeated every 8 h for 32 h starting at 10:00 h on 4 May. People were also advised to report any symptoms to the nursing staff of the wards.

Those who met the case definition were assessed clinically and epidemiological data were collected using a standard questionnaire. Clinical and epidemiological data were also obtained from the records of the Emergency Department. These were combined with data obtained from ward-based surveillance to produce updated time-lines.

Environmental investigation

The IPCT and the hospital management visited the catering facility twice to collect samples, to perform a general inspection, and to review the relevant records. The kitchen was visited immediately after the onset of the outbreak on 4 May and a more extensive audit was conducted on 7 May using a Hazard Analysis and Critical Control Point (HACCP) audit format.

Laboratory methods

Food samples from the kitchen were sent to the Central Public Health Laboratory (CPHL) and subjected to routine qualitative microbiological cultures. In addition, stool samples from all the workers in the kitchen and from people who had gastroenteritis were sent to CPHL and tested for enteric pathogens.

Statistical analysis

The data collected were entered in a database developed for the outbreak and were analyzed using SPSS version 16.0 software.

Results

Epidemiological investigation

From the combined listings, 58 persons were identified as being affected by the gastroenteritis outbreak, of whom 15 were inpatients, 41 were patient relative attendants and 2 were hospital staff members. The majority of cases occurred on paediatric and female obstetric wards, where the number of attending relatives was also concentrated; Table 1 shows distribution of affected persons according to the hospital wards and the attack rates for each ward. The task force decided to send home all those affected, whether attendants or inpatients, who did not need to stay in the hospital. Survey questionnaires were only completed for 50 (86%) of the 58 people affected. Among the 50 with completed questionnaires 40 (80%) were females. Diarrhoea and abdominal pain were present in 36 (72%), diarrhoea alone was present in 9 (18%), abdominal pain alone was present in 2 (4%), and vomiting alone was present in 3 cases (6%). There was no documented fever in any of the cases. Only two patients needed intravenous fluids and none of the attendants required hospitalization. All those affected recovered uneventfully.

The outbreak curve (Fig. 1) summarizes data from the 50 case questionnaires and suggests a point source outbreak. The first symptomatic case presented at 14:00 h on 3 May and the last case was reported on 5 May at around 11:00 h. The onset time for symptoms of most of the cases was between 01:00 h and 05:00 h on 4 May. No cases of secondary transmission were detected.

Table 2 summarizes the risk of developing symptoms related to specific meals taken by the 50 people completing the questionnaire. The incidence of diarrhoea was examined among subgroups based on whether they had taken breakfast, lunch or dinner. Nobody had reported symptoms after having taken breakfast alone. Overall, 44 (88%) had taken varying combinations of lunch, dinner and/or breakfast and 40 (80%) had lunch and dinner. Therefore it was difficult to identify whether lunch or dinner was the specific exposure. However it was found that among this group of 44, while all 3 patients (100%) who had no exposure to dinner developed symptoms, only 4 out of 7 (57%) who were not exposed to lunch had symptoms. Also, although exposure to dinner was more than that of lunch, the timeline of distribution of diarrhoea related to food serving times does indicate that a few cases have reported before the dinner time. Combined with the outbreak curve, showing most cases presenting between 10 and 20 h after lunch at 12:00 h on 3 May, we can consider lunch to be the most likely source of exposure. It is possible that contamination could have also occurred during dinner.

Assuming that lunch was the implicated source of food poisoning, the median (range) incubation period was 15 (2-24) h.

Location in hospital	in hospital Among inpatients		Among attendants			Among all exposed			
	Affected	Total exposed	Attack rate (%)	Affected	Total exposed	Attack rate (%)	Total affected	Total exposed	Attack rate (%)
Paediatric ward 1	0	19	0.0	3	19	15.8	3	38	7.9
Paediatric ward 2	1	18	5.6	11	18	61.1	12	36	33.3
Paediatric ward 3	2	21	9.5	7	21	33.3	9	42	21.4
Paediatric ward 4	0	11	0.0	7	11	63.6	7	22	31.8
Female med ward 1	5	18	27.8	4	4	100.0	9	22	40.9
Female med ward 2	1	22	4.5	1	4	25.0	2	26	7.7
Male med ward 2	3	33	9.1	1	4	25.0	4	37	10.8
Nephro-uro ward	0	21	0.0	1	4	25.0	1	25	4.0
Oncology ward	1	16	6.3	0	4	0.0	1	20	5.0
Special nursing ward	1	13	7.7	2	13	15.4	3	26	11.5
Female surg ward	0	19	0.0	3	4	75.0	3	23	13.0
Maternity ward 4	1	23	4.3	0	4	0.0	1	27	3.7
SCBU	0	27	0.0	1	27	3.7	1	54	1.9
Total	15	261	5.7	41	137	29.9	56	398	14.1

Table 1	The distribution of affected	persons according to 1	the hospital v	wards and the a	ttack rates	for eacl	h ward

Environmental investigation

There were many lapses in the catering procedures. Prepared food was maintained in the danger zone and correct storage procedures were not followed. In addition, there was poor labeling of available food samples.

Cockroaches were found and there were no pest control records. Food items such as biscuits and juices were found hidden behind the bread slicing machine, together with more cockroaches. The refuse conveyer belt was not working, and so there was no immediate removal of refuse from food processing areas. Furthermore, no quarantine facilities were available in the kitchen area for the spoiled and expired food items.

We also found that the tray line staff did not fully cover their nose, and that hand gloves were not used. The staff rest area was untidy and the toilet was dirty. In addition, there were no leg



Figure 1 The outbreak curve from 3 to 4 May 2008, showing the number of cases and the onset time of symptoms of the 50 people for whom a questionnaire was completed. Each column shows the number of patients presenting within an hourly time frame. Assuming lunch distributed at 12:00 h on 3 May as the source of the outbreak, the mean (SD) incubation period is 14.6 (4.6) h.

Timing	Number (%)	Number with diarrhoea (%)		
Breakfast	0	0		
Lunch	1 (2)	1 (100)		
Dinner	5 (10)	2 (40)		
Breakfast + lunch	2 (4)	2 (100)		
Breakfast + dinner	2 (4)	2 (100)		
Lunch + dinner	9 (18)	9 (100)		
Breakfast + lunch + dinner	31 (62)	29 (93.5)		
No meals	0	0		
Total	50	45 (90)		

Table 2 Number and percent of 50-case patients with diarrhoea by meal consumed at Royal Hospital, Oman during May 3–5, 2008.

operated hand wash basins or waste bins in the kitchen area.

Laboratory results

B. cereus was cultured from 35 (57%) of 61 food samples received at the CPHL. All isolates were further tested and found to be toxin producing. The following food items were culture and toxin positive: egg, sewia (a milky dessert), vegetables, meat, chicken, macaroni, noodles, oats, dhal, and foul (beans) masala.

In addition, 19/25 (76%) stool samples from workers in the kitchen and 3/12 (25%) stool samples from patients yielded toxin-producing *B. cereus*.

Discussion

There have been no previous reported food poisoning outbreaks in Oman and this episode caused major public concern. The results of the outbreak investigation confirmed that 58 people had symptoms of gastroenteritis after consuming a meal served from the hospital kitchen served at noon the previous day, and that the causative organism was *B. cereus*.

Although the first case was reported to the Emergency Department at 10:00 h on 4 May, most of those affected developed symptoms earlier on the same day. After the initiation of active surveillance by the task force, many more cases were identified. The onset of the first symptomatic case was 2 h after the implicated lunch meal which had been served around 12:00 h on the previous day. The clinical features were typical of the causative organism and the temporal distribution of cases, with median incubation period of 15 h is consistent with previous reports.

This was the first reported outbreak of food poisoning from Oman and neighboring countries. The number of cases of *B. cereus* foodborne disease is reportedly increasing in industrialized countries [5–8]. The surveillance systems for foodborne disease differ between countries, and so it is difficult to compare data and obtain true incidence estimates. Several factors contribute to underreporting of most outbreaks of foodborne *B. cereus* disease. The clinical course is generally short and mild, so patients rarely seek medical attention, as in this outbreak. When diagnosed, the disease is not always reportable [6].

Different types of food are more commonly associated with the two types of foodborne *B. cereus* disease: the emetic type of disease has often been connected with the consumption of fried and cooked rice [5], pasta, pastry and noodles [3,9]. The diarrhoeal type, which predominated in this outbreak, is commonly associated with proteinaceous foods, sauces and vegetables [10], meat products, soups, puddings and milk products [3,5,7]. The differing patterns of emetic and diarrhoeal disease between countries may reflect the associations of the two types of disease with different food vehicles [5,7].

The majority of the affected cases were reported from the paediatric wards, and this explains why the majority of the affected persons were females, as women usually stay to look after children who are inpatients. In addition, all the patients presented with abdominal pain and diarrhoea. No specific population groups are described as being of special risk for *B. cereus* foodborne disease. However, individuals with lowered stomach acidity may be more susceptible to *B. cereus* diarrhoeal disease [11].

The production of spores is hugely advantageous for *B. cereus*, allowing attachment, as well as survival of heat treatment or other procedures which remove species of vegetative bacteria which could otherwise outgrow *B. cereus* [12]. *B. cereus* spores are not necessarily removed by regular cleaning of surfaces [13,14]. The ability of *B. cereus* to exist in biofilms is most probably important for its persistence in food industry equipment [15], as the biofilm protects spores and vegetative cells from inactivation by sanitizers [16].

Considering the ubiquitous presence and the non-fastidious nature of *B. cereus*, and the resilience of its spores, no type of food with pH > 4.8 can be excluded as a possible vehicle or as representing a risk of food spoilage or foodborne disease [5]. Failure by caterers and consumers to follow basic food preparation rules, i.e. slow or inadequate cooling, storage at ambient temperature or prolonged heat-keeping at <60 °C, may allow growth of *B. cereus* and is commonly part of the story in cases of foodborne disease [6]. All of these faults were observed in the audit of the hospital kitchen.

It was difficult to determine which specific food item was the primary source, as positive cultures were obtained from a wide variety of food items, suggestive of cross contamination between the food items served at lunch [3,5,8]. This was probably exacerbated by the ability of *B. cereus* to live in different food items mentioned above and the poor storage conditions of cooked and uncooked food items together. We were unable to subtype the different isolates of *B. cereus* obtained from food items or from the stools of food handlers, to see if a single clone or multiple clones were present.

The intestines of insects may provide a habitat for *B. cereus*, as spore-forming bacteria have been isolated from the gut of different soil-dwelling arthropod species, in which the bacteria appear to exist in symbiosis [17]. *B. cereus* has also been found in stools of healthy humans [8,10,17,18]. Its ubiquitous low level presence in environments, feed and foods would ensure *B. cereus* a transient presence in the mammalian gut [10]. However, 76% of the 25 catering staff tested had *B. cereus* in their faeces, suggesting widespread lack of adherence to basic hygiene rules, compounded by the poor provision/sitting of appropriate toilet and washbasin facilities on the premises, and providing further sources for the contamination of food.

In order to prevent future outbreaks of food poisoning at the hospital, numerous recommendations were made to the hospital management, most of which related to the catering services of the hospital. There was a clear need to improve the physical condition of the catering facilities and to improve food handling procedures.

This study has several limitations. It was not possible to perform a case control study to identify which meal was the culprit or whether general contamination affected more than one meal. This was compounded by inadequate identification of the food samples that were submitted for culture. Nevertheless, all symptomatic individuals had consumed at least one meal from the kitchen and the results of the environmental audit and the fact that the large majority of food handlers had positive stool cultures strongly implicated the kitchen as the source. The lack of availability of appropriate ribotyping facilities in the CPHL and failure to retain isolates for later testing at a different reference laboratory made it impossible to confirm whether a single or multiple clones of *B. cereus* were responsible.

Institutional outbreaks of food poisoning are rarely reported from the Arabian Peninsula, although community-based outbreaks are common, especially during large aggregations of people such as the Hajj pilgrimage [19,20]. Most food poisoning outbreaks reported from the neighboring Kingdom of Saudi Arabia have been attributed to salmonellosis [19,21] with relatively few attributed to viruses or pre-formed bacterial toxins.

Many catering facilities in the region are manned by expatriate workers, typically from the Indian Sub-continent, and occupational health screening for such staff usually focuses on excluding the carriage of pathogenic parasites [22,23], which are of questionable importance. The need for continued education of such staff about food hygiene and for the enforcement of relevant regulations has been emphasized recently in the region [24–26].

As a result of this outbreak, major refurbishments were made in the kitchen, food handlers underwent further education (ongoing) and food hygiene regulations are now strictly controlled. The hospital response to media enquiries has been modified. Outbreak investigation protocols have been improved, including the need to retain microbiological samples for later testing by external reference laboratories.

Conclusion

An outbreak of *B. cereus* food poisoning at the Royal Hospital, Oman, affected 58 inpatients and their attendants in May 2008. Epidemiological evidence incriminated one meal prepared in the hospital kitchen, but it was not possible to establish which specific food item was the cause of the outbreak. This outbreak was the result of many breaches of basic hygiene regulations in a hospital kitchen, despite a recent external quality assurance inspection. A national scandal ensued, resulting in the lack of confidence in the main tertiary hospital for the country, although fortunately no patients came to any long-term harm. Following this, many improvements have been made to physical facilities in the kitchen area and to the regular education of kitchen staff, with enforcement of basic hygiene rules, and no further outbreaks or sporadic cases have occurred.

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Competing interest

None declared.

Ethical approval

Not required.

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References

- Bottone EJ. Bacillus cereus, a volatile human pathogen. Clin Microbiol Rev 2010;23:382–98.
- [2] Dohmae S, Okubo T, Higuchi W, Takano T, Isobe H, Baranovich T, et al. Bacillus cereus nosocomial infection from reused towels in Japan. J Hosp Infect 2008;69:361–7.
- [3] Granum PE. Bacillus cereus. In: Doyle MP, Beuchat LR, editors. Food microbiology: fundamentals and frontiers. Washington, DC: ASM Press; 2007. p. 445–55.
- [4] Schmidt K. WHO surveillance program for control of foodborne infections and intoxications in Europe. Berlin, Germany: FAO/WHO Collaborating Centre for Research and Training in Food Hygiene and Zoonoses; 2001 [Report No.: 7].
- [5] Gilbert RJ, Kramer JM. Bacillus cereus food poisoning. In: Cliver DC, Cochrane BA, editors. Progress in food safety (proceedings of symposium). Madison, WI: Food Reasearch Institute, University of Wisconsin-Madison; 1986. p. 85–93.
- [6] Stenfors Arnesen LP, Fagerlund A, Granum PE. From soil to gut: Bacillus cereus and its food poisoning toxins. FEMS Microbiol Rev 2008;32:579–606.
- [7] Kotiranta A, Lounatmaa K, Haapasalo M. Epidemiology and pathogenesis of Bacillus cereus infections. Microbes Infect 2000;2:189–98.
- [8] Johnson KM. Bacillus cereus food-borne illness an update. J Food Prot 1984;47:145–53.
- [9] Schoeni JL, Wong AC. Bacillus cereus food poisoning and its toxins. J Food Prot 2005;68:636–48.

- [10] Kramer JM, Gilbert RJ. Bacillus cereus and other bacillus species. In: Doyle MP, editor. Foodborne bacterial pathogens. New York: Marcel Dekker; 1989. p. 21–70.
- [11] Clavel T, Carlin F, Lairon D, Nguyen-The C, Schmitt P. Survival of Bacillus cereus spores and vegetative cells in acid media simulating human stomach. J Appl Microbiol 2004;97:214–9.
- [12] Faille C, Tauveron G, Le Gentil-Lelievre C, Slomianny C. Occurrence of Bacillus cereus spores with a damaged exosporium: consequences on the spore adhesion on surfaces of food processing lines. J Food Prot 2007;70:2346–53.
- [13] Faille C, Jullien C, Fontaine F, Bellon-Fontaine MN, Slomianny C, Benezech T. Adhesion of Bacillus spores and Escherichia coli cells to inert surfaces: role of surface hydrophobicity. Can J Microbiol 2002;48:728–38.
- [14] Andersson A, Ronner U, Granum PE. What problems does the food industry have with the spore-forming pathogens Bacillus cereus and Clostridium perfringens? Int J Food Microbiol 1995;28:145–55.
- [15] Wijman JG, de Leeuw PP, Moezelaar R, Zwietering MH, Abee T. Air-liquid interface biofilms of Bacillus cereus: formation, sporulation, and dispersion. Appl Environ Microbiol 2007;73:1481–8.
- [16] Ryu JH, Beuchat LR. Biofilm formation and sporulation by Bacillus cereus on a stainless steel surface and subsequent resistance of vegetative cells and spores to chlorine, chlorine dioxide, and a peroxyacetic acid-based sanitizer. J Food Prot 2005;68:2614–22.
- [17] Jensen GB, Hansen BM, Eilenberg J, Mahillon J. The hidden lifestyles of Bacillus cereus and relatives. Environ Microbiol 2003;5:631–40.
- [18] Yea CL, Lee CL, Pan TM, Horng CB. Isolation of Bacillus cereus in the feces of healthy adults in Taipei City. Zhonghua Min Guo Wei Sheng Wu Ji Mian Yi Xue Za Zhi 1994;27:148–51.
- [19] Al-Mazrou YY. Food poisoning in Saudi Arabia. Potential for prevention? Saudi Med J 2004;25:11-4.
- [20] Ahmed QA, Arabi YM, Memish ZA. Health risks at the Hajj. Lancet 2006;367:1008–15.
- [21] Al-Ahmadi KS, El Bushra HE, Al-Zahrani AS. An outbreak of food poisoning associated with restaurant-made mayonnaise in Abha, Saudi Arabia. J Diarrhoeal Dis Res 1998;16:201–4.
- [22] Abu-Madi MA, Behnke JM, Ismail A. Patterns of infection with intestinal parasites in Qatar among food handlers and housemaids from different geographical regions of origin. Acta Trop 2008;106:213–20.
- [23] Gunduz T, Limoncu ME, Cumen S, Ari A, Serdag E, Tay Z. The prevalence of intestinal parasites and nasal S. aureus carriage among food handlers. J Environ Health 2008;70(64-5):67.
- [24] Askarian M, Kabir G, Aminbaig M, Memish ZA, Jafari P. Knowledge, attitudes, and practices of food service staff regarding food hygiene in Shiraz, Iran. Infect Control Hosp Epidemiol 2004;25:16–20.
- [25] El DH, Salem E, Fawzi M, Abdel AM. Safety of patient meals in 2 hospitals in Alexandria, Egypt before and after training of food handlers. East Mediterr Health J 2008;14:941-52.
- [26] Plus NHS, Faculty of Occupational Medicine. Infected food handlers: occupational aspects of management. London: Royal College of Physicians; 2008.

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