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Outbreak of *Clostridium perfringens* food poisoning

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Summary: An outbreak of diarrhoea occurred at a 647-bedded long-stay hospital from 11 to 14 June 1989. Fifty-eight elderly residents developed symptoms and there were two deaths. The organism was identified as *Clostridium perfringens* type A, serotype TW23. The source of the outbreak was found to be inadequately reheated minced beef served at lunchtime on 11 June. The reason why only 4 of the possible 16 wards receiving minced beef were affected was thought to be due to the division of the batch of minced beef at a late stage in the preparation process. We conclude that there is a need for effective bowel and nutrition policies and that these are high priorities for audit.

Keywords: *Clostridium perfringens*; outbreak; food poisoning.

Introduction

Clostridium perfringens has only recently been recognized as an important cause of foodborne disease. This may be because morbidity is seldom severe and the case fatality rate is as low as 0·07%. It does, however, contribute significantly to outbreaks of food poisoning, estimates of incidence varying from 10 to 20%.[1]

Hospital outbreaks of *C. perfringens* are very common. Of 48 hospital outbreaks notified in Scotland from 1978–1987, 25 (51%) were caused by *C. perfringens*.[2] The organism multiplies optimally at 37–41°C, and although there is no seasonal prevalence, higher numbers of outbreaks have been observed to occur in the summer months.[3] The outbreak reported here occurred in June 1989 at the height of a heatwave, when outside temperatures were in excess of 32°C. Establishing an outbreak of *C. perfringens* can be difficult, as the organism is frequently found as a commensal in the gut, especially in residents of long-stay institutions.[4] While the human excretor role is not thought to be important, the organism

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is ubiquitous—hence the difficulty in establishing the source of the infection. For this reason, in addition to serotyping the organism to confirm the extent of the outbreak, we also carried out a case control study and a validation exercise in order to establish the source of the outbreak.

Investigation of the outbreak

The outbreak occurred in a 647-bedded long-stay psychogeriatric hospital in a district in Greater London. It is a large, sprawling Victorian institution with extensive grounds and scattered outlying villas. The delivery of food around its perimeter is not easy. Following a previous outbreak of *C. perfringens* food poisoning in 1987 there were extensive improvements to the food transportation system. The hospital kitchens were built at the same time as the main hospital and are difficult to maintain, despite extensive renovation. Unlike many of its Victorian counterparts this hospital has no closing date, although the bed numbers have been reduced in line with current policy for care in the community.

Epidemiological study

An incident room was set up. All new cases were charted by place and time of onset of symptoms. A menu of all meals eaten over the 2 days prior to the outbreak was compiled and checked with catering staff. A food questionnaire of meals eaten was completed by staff for all patients who presented as cases and one ward was used for a case control study. The questionnaire requested details of meals eaten by the cases, including the two deaths, in the 48 h prior to the outbreak, and the date and time of onset of symptoms.

Case definition. Diarrhoea of onset on or after 11 June (up to and including 13 June for the purpose of exclusion).

Controls. The same questionnaire was completed by staff on the unaffected residents (controls) on the ward with the largest number of cases ($N=17$). This served as a control group for later analysis.

Validation. Because of problems with recall, staff on the control ward were also asked on a separate occasion whether residents usually ate a 'normal' or 'soft' diet and these responses were matched against the questionnaires.

Environmental investigation

The hospital kitchens were inspected by the Environmental Health Officer but no cooked food samples were available for examination.

Microbiological investigation

(i) Faecal samples were requested from all cases and in addition specimens were collected from the two deceased patients undergoing *post mortem*; in one case, a pre-mortem specimen had been taken.

(ii) A specimen of raw minced beef was also cultured selectively for intestinal pathogens.

Interviews with catering staff

Catering staff who were on duty prior to the outbreak were interviewed extensively about the preparation of food in the 48 h prior to the outbreak.

Analysis of food questionnaire

Time and date of onset of symptoms. From Figure 1 it can be seen that there were two peaks of incidence. The initial large peak corresponds with the first cases presenting at about 6.00 p.m. on 11 June and the majority occurring between midnight and 6.00 a.m. on 12 June. The second peak presented 48 h after the initial outbreak and appeared to affect two further wards (E, F) plus the original ward (A), and affected eight patients.

Analysis of meals eaten. All 49 cases presenting in the 24 h period between 6.00 p.m. on 11 June to 6.00 a.m. on 12 June had eaten the 'soft' minced-beef meal for lunch on 11 June. However, of the eight cases presenting in the later peak on 13 June, only two had eaten minced beef.

Case control study. There were 32 residents on the ward used in the case control study; 15 cases and 17 unaffected patients who were used as controls. The results of the food questionnaire and validation study are presented in Table I.

The food questionnaire showed that of the 20 patients who appeared to have eaten the minced beef meal there were only 15 cases. However, the

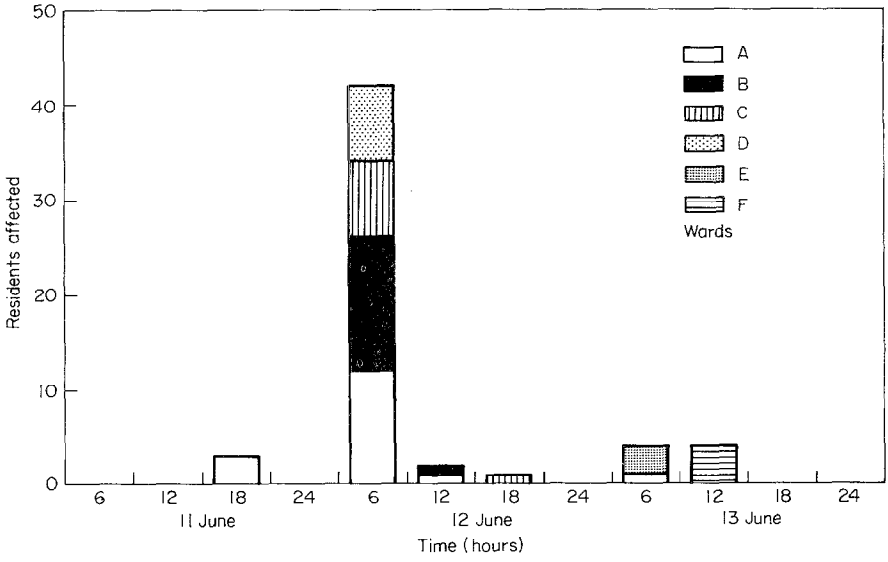

Figure 1. Epidemic curve of *C. perfringens* outbreak, 11–13 June.

Table I. *Validation exercise comparing food questionnaires with staff's knowledge of residents' regular daily diet*

Validation questionnaire	Minced beef 'Soft diet'	Roast beef 'Normal diet'	Total
Minced beef	17	3	20
Roast beef	0	12	12
Total	17	15	32

validation study subsequently showed that three of the five unaffected patients (controls) said to have eaten minced beef would in fact have eaten the normal diet. The remaining two patients (controls) only ate very small portions suggesting a possible dose response.

Case attack rate. Because of later problems establishing a true denominator, a case attack rate could only be established for the patients on the control ward. This was 15/17 (88%).

Case fatality rate. The case fatality rate was two deaths out of 49 cases (4%).

Microbiological results

From Table II it can be seen that only 34 stool specimens were received from the total of 58 cases (59%). Culture of the specimens yielded enterotoxin-producing strains of *C. perfringens* from 26 (76%). Serotyping confirmed that the outbreak was due to *C. perfringens* type A serotype TW23. The outbreak was confined to the four wards presenting in the first 24 h of the outbreak. Of the eight cases presenting 48 h after the onset of the outbreak, five specimens were received. The two specimens positive for *C. perfringens* were of serotypes 39 and 9, and therefore not part of the initial outbreak.

Clostridium perfringens was not isolated from the remains of the raw minced beef.

Table II. *Microbiological analysis of faecal specimens*

Ward	No. of patients affected	No. of specimens received	No. positive for *C. perfringens* (%)	Type A serotype
A	17	10	8 (80)	TW23
B	15	10	10 (100)	TW23
C	9	4	4 (100)	TW23
D	8	5	2 (40)	TW23
E	4	2	0 (0)	—
F	4	3	2 (75)	39, 9
G	1	0	0 (0)	—
Total	58	34	26 (76)	

Post-mortem findings
At post mortem it was discovered that both patients had evidence of a grossly distended colon and rectum combined with longstanding severe faecal impaction. In addition one had a medical history of ischaemic heart disease and the other emphysema.

Environmental inspection
The Environmental Health Officer failed the kitchen premises on 113 accounts. The main findings related to the considerable accumulation of grime and dirt in the kitchen and poor maintenance and food handling.

Interviews with catering staff
Interviews with the kitchen staff who were on duty prior to the outbreak revealed errors in food preparation and food handling. The mince served for the lunchtime meal on 11 June had been cooked two days previously (in direct contravention of the Department of Health guidelines[5]). There had also been problems with inadequate chilling and storage. The refrigerators were above recommended temperatures. Furthermore it was clearly established that the mince had not been adequately reheated and had provided ideal conditions for germination of the spores to a vegetative state, leading to multiplication of the organism. However, although when we reviewed the menu sheets it appeared that 16 wards would have received, and 118 patients should have eaten, minced beef on the day in question, it was apparent that only four wards and 49 patients were affected. We again traced the food preparation, looking at the menu sheet that the cook had worked from on 11 June. From the sheet two facts emerged: (i) the affected wards were on the top half of the sheet; (ii) affected patients appeared to come from the wards receiving the largest number of portions.

Discussion

In view of the findings from our investigation we propose that the following course of events led to the outbreak. On the day of the outbreak, the minced beef was inadequately reheated in the steamer. It was then transferred, still in the deep metal container, to the hot stove where it continued cooking. The mince was divided into two batches. The first batch corresponded with the first half of the menu sheet and the second batch with the remaining wards. The mince for each batch was apportioned into various sized containers. The wards receiving eight or more portions received their portions in large deep metal trays. However, wards receiving smaller portions received their meals in small individually packaged tin foil trays.

The inadequately reheated mince now contained the organism *C. perfringens* in the vegetative state, where it could freely multiply at 31–41°C. The organism was distributed into the deep metal trays in the first batch; the metal trays were placed in heated trolleys. However, they never

reached an adequate temperature, resulting in optimal conditions for growth of the organism. Those mince portions in the small individual trays did achieve adequate temperature in the heated trolleys, which are maintained at 80°C. Over the next few hours between 10.00 and 12.30 a.m. the organism did not survive in the small individual trays.

Meanwhile the second batch of mince, still on the hot stove, continued to cook to the proper temperature, thus reducing the concentration of the organism in its vegetative state. It is known that the organism is ingested in the vegetative state, and undergoes sporulation in the intestine where it releases enterotoxin, causing gastrointestinal symptoms.[1]

This investigation was made easier by having a 'captive' resident population. Despite problems with recall bias and staff having to administer questionnaires, we achieved a 100% response rate. The restricted daily menu limited recall errors by staff and made the validation study possible.

The time of onset of symptoms, together with generally mild symptoms of diarrhoea, are consistent with a *C. perfringens* outbreak.[1] The organism *C. perfringens* is a frequent cause of institutional outbreaks of food poisoning, although the incidence has declined markedly in the last decade possibly due to the introduction of cook–chill and increased awareness of contributing factors. Nevertheless, *C. perfringens* still accounted for more than 50% of hospital outbreaks in Scotland between 1978 and 1987, the majority of which occurred in long-stay institutions which are also at greater risk of repeat outbreaks.[2]

It can be difficult to identify the true extent of an outbreak, due to the ubiquitous nature of the organism. Many strains are found not only in the gastrointestinal tract of animals and humans, but also soil and dried foods.[1] It is not uncommon for healthy residents, particularly in long stay institutions, to excrete *C. perfringens*. Studies have shown that up to 30% of the hospitalized elderly may carry the organism.[4]

For this reason the technique of serotyping and enterotoxin analysis is particularly useful in epidemiological analysis to distinguish cases, as patients may excrete several strains of the organism.

This outbreak was caused by type A, serotype TW23. Two other strains were identified in the investigation and belonged to serotypes 39 and 9. These may have been pathogens or simply commensals in the gut, but were not part of the main outbreak.

The minced beef meal was responsible for the outbreak. Minced meat has been implicated previously in *C. perfringens* outbreaks. In seven of the 64 *C. perfringens* outbreaks occurring in 1985 the source was minced beef served in hospitals or other institutions.[6] In this study the original sample of raw minced beef was culture negative, suggesting that contamination occurred during the preparation or cooking process. The Department of Health no longer recommends keeping cooked food samples; in this instance keeping samples may not have been helpful as it would depend on which stage in the cooking process the sample was saved. It does seem that the multiplication

of the organism continued after the division of the first batch of minced meat diets.

A similar time trend has been observed previously during a large outbreak of *C. perfringens*, affecting over 600 employees in a Connecticut factory. Stratified analysis indicated that gravy was the food responsible, and that there was a decreasing attack rate over serving periods with the lowest attack rates occurring in those exposed to gravy which had been reheated for the longest time when the organism was at a lower concentration.[7]

Both our cases and the Connecticut study demonstrate that inadequate reheating was responsible for high attack rates, but that continued cooking to adequate temperatures diminished the risk of food poisoning.

The detailed interviews with the kitchen staff who were on duty over the weekend prior to the outbreak, revealed widespread evidence of bad food practice and poor food handling. The reasons behind this have been examined, and are the subject of a separate report.[8]

In our investigation the case fatality rate was 4%. This compares with 0·07% for England and Wales in the 12-year period between 1969 and 1980.[1] Of the 899 patients affected by *C. perfringens* in hospital outbreaks in Scotland between 1978 and 1987 the case fatality rate was 0·3%. This finding would suggest our proposition that the relevant population in our study were frail and more susceptible than the general population. Furthermore the findings of chronic faecal impaction at post mortem in the patients who died raises medical and nursing concerns. It was evident from the deceased patients' nursing and medical notes that their clinical condition and their nutritional state had not been regularly recorded in the year prior to their decease.

Since long-stay residents may be particularly vulnerable to food poisoning, adequate monitoring of diarrhoea or constipation is essential. Medical and nursing staff responsible for the care of long-stay residents should review and implement nutrition and bowel care policies. This must involve close liaison with district dieticians and catering staff and also consideration of the contribution of geriatricians.

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