An outbreak of foodborne botulism associated with contaminated hazelnut yoghurt

M. O'MAHONY¹, E. MITCHELL^{1*}, R. J. GILBERT², D. N. HUTCHINSON³, N. T. BEGG¹, J. C. RODHOUSE² and J. E. MORRIS⁴

¹PHLS Communicable Disease Surveillance Centre, 61, Colindale Avenue, London NW9 5EQ ²PHLS Food Hygiene Laboratory ³PHLS Preston Laboratory ⁴Blackpool, Wyre and Fylde Health Authority

(Accepted 19 December 1989)

SUMMARY

The largest recorded outbreak of foodborne botulism in the United Kingdom occurred in June 1989. A total of 27 patients was affected; one patient died. Twenty-five of the patients had eaten one brand of hazelnut yoghurt in the week before the onset of symptoms. This yoghurt contained hazelnut conserve sweetened with aspartame rather than sugar. *Clostridium botulinum* type B toxin was detected in a blown can of hazelnut conserve, opened and unopened cartons of hazelnut yoghurt, and one faecal specimen. *Cl. botulinum* type B was subsequently cultured from both opened and unopened cartons of the hazelnut yoghurt and from one faecal specimen. Investigations indicated that the processing of the conserve was inadequate to destroy *Cl. botulinum* spores. Control measures included the cessation of all yoghurt production by the implicated producer, the withdrawal of the firm's yoghurts from sale, the recall of cans of the hazelnut yoghurts.

INTRODUCTION

Foodborne botulism in man is rare in the United Kingdom. There have been only nine incidents reported since 1922, and in three of these only one person was affected. In the largest of these outbreaks, associated with Loch Maree in 1922, eight people died after eating duck paste [1]. Five of the other eight incidents involved home prepared dishes [2–8], one was associated with pickled fish from Mauritius [9], one with canned Alaskan salmon [10], and the remaining one with

^{*} Author for correspondence: Dr Elizabeth Mitchell, Communicable Disease Surveillance Centre, 61 Colindale Avenue, London, NW9 5EQ, UK.

The following were members of the field investigation team: Mr R. Ridley, Mr I. Almond, Mr D. Tilleray, Blackpool BC Environmental Health Department, Mr I. Dowty, Wyre BC Environmental Health Department, and Mr K. Garritty, Preston BC Environmental Health Department; Dr S. Payne, Dr M. Catchpole, Dr M. Evans, Dr S. Sethi, and Miss J. Bruce, PHLS Communicable Disease Surveillance Centre.

M. O'MAHONY AND OTHERS

an airline meal [11]. We describe the epidemiological and microbiological investigations of a large outbreak of foodborne botulism which occurred in the North West of England and Wales in June 1989; the clinical features have been described elsewhere [12].

THE OUTBREAK

On Thursday, 8 June 1989, a 47-year-old female in a Blackpool hospital was reported to have suspected botulism. Her son was in the Intensive Therapy Unit (ITU) in a Preston hospital with a diagnosis of Guillain-Barré syndrome. In the same ITU there was a second patient who was also thought to have this syndrome. The next day, two further patients in the Blackpool hospital and two children, a brother and sister, in a Manchester hospital were reported to have suspected botulism. Four of the patients were receiving positive pressure ventilation. An investigation was commenced to determine the cause and the source of the outbreak. On Saturday, 10 June, the diagnosis of suspected botulism was made in a third child in the Manchester hospital.

Preliminary investigations showed that seven of the eight patients had eaten one brand of hazelnut yoghurt, made by Producer 1, in the week before the onset of symptoms: the remaining patient was too ill to be interviewed. No other food item had been eaten in common by all of the patients. Late on Sunday, 11 June, the Department of Health (DoH) was informed of the association between illness and the consumption of hazelnut yoghurt and it was decided to stop the production of all Producer 1's yoghurts and to withdraw these products from sale, and the public were advised to avoid the consumption of this brand of hazelnut yoghurt. On 12 June, another firm (Producer 2), which used the same brand of hazelnut conserve in its yoghurt, also stopped production and the DoH broadened its advice to the general public to avoid the consumption of any brand of hazelnut yoghurt. Following the introduction of control measures, a further 19 patients were identified by case searching and a descriptive study was carried out.

METHODS

A case of suspected botulism was defined as a person with at least three of the following clinical features, dysarthria, dysphagia, diplopia, bilateral weakness of the limbs and weakness of the respiratory muscles, and who did not have fever on presentation or sensory symptoms. A case search was carried out by requesting local medical officers for environmental health to report any cases of suspected botulism. Those people identified by the case search were interviewed by use of a semi-structured but detailed questionnaire. Information was requested on symptoms and the date of onset of illness. In addition, information was sought on foods eaten in the week before onset of symptoms, with particular emphasis given to foods implicated in previous outbreaks of botulism, such as canned, bottled or smoked products.

Serum and faecal specimens were obtained from suspected cases and sent to the Public Health Laboratory Service (PHLS) Food Hygiene Laboratory (FHL) for the detection of *Clostridium botulinum* toxin. Cans of implicated hazelnut conserve and opened and unopened cartons of Producer 1's yoghurt were sent to the FHL.

390

Botulism associated with hazelnut yogurt 391

Two samples of another brand of yoghurt, made by Producer 3, were forwarded for examination. Sera, extracts of foods, and faecal extracts were injected into mice. Protection tests with polyvalent and monovalent antisera were carried out on extracts of the hazelnut conserve from the blown can. Faecal specimens and food samples were also cultured for *Cl. botulinum* and isolates were sent for confirmation to the PHLS Anaerobe Reference Unit, Luton. Sera and food samples were also tested using an amplified ELISA procedure for the immunological detection of toxin at the PHLS Centre for Applied Microbiology and Research (CAMR), Porton Down [13]. The concentration of toxin in conserve and yoghurt was calculated using mouse lethal dose (MLD) determinations.

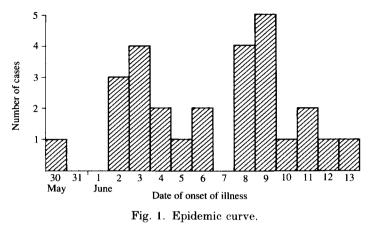
The premises of Producer 1 were inspected and information obtained on the method of yoghurt production. In turn, the premises of the conserve manufacturer were inspected. The premises of two other yoghurt producers, Producers 2 and 3, were also inspected.

RESULTS

A total of 27 patients was identified, of whom 10 were males and 17 were females with an age range between 14 months and 74 years, median 29 years. One patient, a 74-year-old female, died with an aspiration pneumonia. Twenty-five of the 27 patients lived in North West England and the remaining two lived in North Wales. Twenty-six patients were admitted to hospital; one patient with mild symptoms did not consult her general practitioner at the onset of her illness but was examined later by a consultant physician. The dates of onset of symptoms ranged between 30 May and 13 June (Fig. 1).

Twenty-five of the 27 patients had eaten Producer 1's hazelnut yoghurt. The dates of consumption were known for 24 and they had eaten the yoghurt between 26 May and 10 June. The interval between consumption and the onset of symptoms was between 2 h and 5 days, with a median of 1 day. The amount of yoghurt consumed varied from a couple of spoonfuls to three cartons. Some of those affected commented that the yoghurt had a different taste from usual and that the nuts tasted bitter. The two remaining patients had eaten Producer 3's yoghurt and both ate it on 6 June.

A total of 21 cans of hazelnut conserve were examined and Cl. botulinum type B toxin was detected by the two methods in conserve from one sealed but badly blown can obtained from Producer 1's premises. The pH of the hazelnut conserve in 15 of 17 cans was found to be between 5.0 and 5.5, in the other two the pH was 4.5 and 4.7 respectively. Both of two opened cartons recovered from patients' homes, and all of 15 unopened cartons of Producer 1's hazelnut yoghurt yielded type B toxin by the mouse bioassay procedure: all 17 positive cartons had a sell by date of 13 June 1989. *Cl. botulinum* type B toxin was detected in faeces from one patient by the mouse bioassay test, but faecal specimens from 8 others and sera from 23 patients were all negative. The levels of type B toxin in samples of the hazelnut conserve were 600–1800 and 14–30 MLD/ml in the contaminated yoghurt. In addition, *Cl. botulinum* type B (proteolytic) was cultured from the faeces of one patient, in whose specimen toxin had been detected, and from opened and unopened cartons of Producer 1's hazelnut yoghurt. The results of tests carried out on two of Producer 3's yoghurts were negative.



The conserve manufacturer prepared the hazelnut conserve from a mixture of pre-roasted hazelnuts, water, starch, and other ingredients, which was heated in a steam jacketed vat with a half ton capacity to a temperature of 90 °C for 10 min. The mixture was pumped into metal cans which were closed at the top using a manually operated seamer and then placed in a retort of boiling water for a minimum of 20 min. Most of the hazelnut conserve manufactured contained sugar. Seventy-six cans of conserve sweetened with aspartame rather than sugar were manufactured in July 1988, 36 cans from this consignment were received by Producer 1 in November 1988 and stored at ambient temperature. A further 36 cans had been delivered to another firm, Producer 2, in July and August, 1988. Two of the remaining four cans were still held by the manufacturer and it was believed that two had been used for testing purposes some time previously. Producer 3 had received a supply of this manufacturer's hazelnut conserve on 28 June 1988, which they believed had been used soon after delivery, but records were not available to confirm this. Since that time they had obtained supplies of hazelnut conserve from another firm. Customers reported to the conserve manufacturer that cans of the aspartame sweetened hazelnut preparation had blown and, following this, in October 1988, potassium sorbate was introduced into the mixture, in an attempt to control yeasts.

The premises of Producer 1 were inspected and were of adequate standard for the production of yoghurt. The yoghurt was produced from a mixture of pasteurized milk, skimmed milk powder and starch, which was heated to 82 °C, had sugar added and held for 30 min. The mixture was cooled, pumped to an inoculating tank, starter culture added and mixed for 20 min, following which it was poured into ten gallon churns and placed in an incubator for 2–3 h until 'set'. Hazelnut conserve was then mixed with the yoghurt, before dispensing the product into 360 cartons, each with a 'sell-by' date 25 days after production. Advice was given on the cartons that the yoghurt should be refrigerated and consumed within 2 days of purchase. The standards of hygiene at the premises of Producer 2 and Producer 3 were satisfactory.

DISCUSSION

This incident of suspected foodborne botulism is the largest recorded outbreak in the UK. The largest previously reported outbreak was the Loch Maree incident in 1922 [1], since then only 21 patients with this disease have been recorded [2–11]. Only one of these episodes, in which the implicated food was a home-made meat pie, was due to *Cl. botulinum* type B toxin [7].

Although almost half of the patients were admitted to ITUs and a third needed positive pressure ventilation, only one patient died. Case fatality rates have decreased significantly in recent decades, which is probably related to the improvements in clinical management and the availability of antitoxin [13]. In this outbreak, several of the later cases had mild symptoms and the diagnosis of suspected botulism was not made on initial presentation [12]. It has been suggested in other reports that type B botulism progresses more slowly and is less severe than type A [14, 15]. *Cl. botulinum* type B toxin was detected in faeces from one patient but not from patients' sera: in an outbreak of type B botulism associated with chopped garlic, toxaemia was only identified in 3 of 36 patients [15].

The epidemiological study suggested an association between illness and the consumption of Producer 1's hazelnut yoghurt, which was confirmed by the results of the microbiological investigations. Type B toxin was detected in all 17 cartons with a sell by date of 13 June. This suggested that all 360 cartons of yoghurt produced on 19 May had the potential to cause botulism and that there may have been cases which were undetected. However, prompt identification of the food vehicle may have prevented some cases.

This outbreak provided an unique opportunity to determine the level of Cl. botulinum toxin in a food incriminated in an outbreak of foodborne botulism. The levels were 1750–3750 MLD per 125 g carton of yoghurt.

The detection of *Cl. botulinum* type B toxin in one can of conserve provided evidence that the yoghurt was contaminated by this ingredient. Enquiries into the methods used in the preparation of the conserve showed that there was insufficient heat used in the manufacturing process to destroy spores of *Cl. botulinum*. The low acidity of the conserve would have allowed the growth of *Cl. botulinum* and the production of toxin. Other fruit mixtures produced by the company had much lower pHs and would be less conducive to growth and production of toxin. Outbreaks of botulism from commercial canned foods are rare and are usually associated with low-acid foods (pH greater than 4.6) [14]. Only one incident linked to the consumption of nuts has been reported, and this was an outbreak of type A foodborne botulism associated with commercially preserved peanuts in Taiwan in 1986 [16].

Although most patients ate Producer 1's yoghurt, two had only consumed Producer 3's yoghurt suggesting a second source of contaminated yoghurt. There are three possible explanations for the association. One, the patients had eaten Producer 1's hazelnut yoghurt but could not remember the occasion. The patients were interviewed several times and did not waiver in their story. Two, the diagnosis in these two patients was incorrect, however the attending physicians made a firm clinical diagnosis and both had at least one other patient with

M. O'MAHONY AND OTHERS

botulism under their care. Three, not all Producer 3's conserve was used in 1988 but some remained which was added to yoghurt made by the firm in May or June 1989; this was denied by the firm. Lastly, some other food vehicle may have been responsible.

The severity of the illness meant that it was necessary to introduce control measures immediately and therefore an analytical epidemiological study was not carried out. The control measures were successful as there were no cases with date of onset of illness after 13 June. Following the control of the outbreak, on 26 June, the DoH lifted the warning to the public not to eat hazelnut yoghurts [17]. The DoH have produced guidance for Environmental Health Departments on the prevention of botulism, which recommend that they assess the safety of all types of food processing in their districts where foods are packaged anaerobically [18].

This outbreak demonstrates the value of the rapid establishment of good communications between all those concerned with the investigation of suspected foodborne botulism. If the diagnosis is suspected the local microbiologist and consultant in control of communicable disease should be contacted as a matter of urgency. In the UK the PHLS Communicable Disease Surveillance Centre and the PHLS Food Hygiene Laboratory, Colindale should also be informed. Arrangements should be made for the urgent examination of serum and faecal specimens as well as food samples.

ACKNOWLEDGEMENTS

We thank the staff of Blackpool, Wyre and Fylde Health Authority who assisted the investigating team, and in particular we would like to thank Dr D. Lewis, Consultant Microbiologist, Dr A. Irving, DMO, Mr Carter, DGM, and Mr G. Wildish, UGM. We gratefully acknowledge the help of the patients and their families, and the consultant physicians and other clinical staff at Blackpool Victoria Hospital, Booth Hall Hospital, Bury General Hospital, Glan Clwyd Hospital, Monsall Hospital, Oldham General Hospital, Royal Preston Hospital, and Walton Hospital.

We thank Miss C. Haugh, Mr J. Kramer and Miss M. Kendall, Food Hygiene Laboratory, Dr C. Shone, PHLS Centre for Applied Microbiology and Research, and Dr A. T. Willis and Mr J. Brazier, PHLS Anaerobe Reference Unit for their expert assistance, as well as Mr. M. Jacob and Mr N. Kingcott, DoH for information about the production processes.

REFERENCES

- 1. Leighton GR. Report of the circumstances attending the deaths of eight persons at Loch Maree, Ross-shire, Official Report to the Scottish Board of Health. Edinburgh: His Majesty's Stationary Office, 1923.
- 2. Nash G, Fox EP. Two cases of botulism. Br Med J 1932; 2: 966-7.
- 3. Nash WG. A case of botulism: recovery. Br Med J 1934; 2: 1199.
- 4. Templeton WL. Two fatal cases of botulism. Br Med J 1935; 2: 500-1.
- 5. Lane CR, Jones-Davies TE. A case of botulism with a note on the bacteriological examination of the suspected food. Lancet 1935; ii: 717-8.
- 6. Kitkat CW. A fatal case of botulism. Br Med J 1935; 2: 580.
- 7. Aitken RS, Barling B, Miles AA. A case of botulism. Lancet 1936; ii: 780-4.

394

395

- 8. Ministry of Health. Report of the Ministry of Health for the year ended 31st March 1949. London: His Majesty's Stationary Office, 1950: 90-1.
- 9. Mackay-Scollay EM. Two cases of botulism. J Path Bact 1958; 75: 482-5.
- 10. Ball AP, Hopkinson RB, Farrell ID, et al. Human botulism caused by *Clostridium* botulinum type E: the Birmingham outbreak. Quart J Med 1979; **191**: 473-91.
- Public Health Laboratory Service. Botulism associated with an in-flight meal. CDR 1987; 87/42: 1.
- Critchley EMR, Hayes PJ, Isaacs PET. Outbreak of botulism in north west England and Wales, June 1989. Lancet 1989; ii: 849-53.
- Shone C, Wilton-Smith P, Appleton N, et al. Monoclonal antibody-based immunoassay for type A *Clostridium botulinum* toxin is comparable to the mouse bioassay. Appl Env Microbiol 1985; 50: 63-7.
- 14. Hauschild AHW. Clostridium botulinum. In Doyle MP. (ed) Foodborne bacterial pathogens. New York: Marcel Dekker Inc; 1989: 111-89.
- 15. St Louis ME, Peck SHS, Bowering D, et al. Botulism from chopped garlic: delayed recognition of a major outbreak. Ann Int Med 1988; 108: 363-8.
- Chou JH, Hwang PH, Malison MD. An outbreak of type A foodborne botulism in Taiwan due to commercially preserved peanuts. Int J Epidemiol 1988; 17: 899-902.
- 17. Department of Health Botulism: all-clear for hazelnut yoghurts. (Press Release) 1989, 26 June.
- Department of Health. Guidance for Environmental Health Departments on the prevention of botulism. EL(89)P 145 1989 29 August.