

Original Article

***Clostridium perfringens* Foodborne Outbreak due to Braised Chop Suey Supplied by Chafing Dish**

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On February 13, 2002, a public health center in Hiroshima Prefecture, Japan, was notified that many individuals living at the Japan Maritime Self-Defence Force base had symptoms resembling those of food poisoning. Self-administered questionnaires requesting information regarding meal consumption and symptoms were distributed to all 281 members at the base. A case of the illness was defined as a member who had had watery or mucousy stool, or loose stool with abdominal cramps, more than twice a day after consuming dinner on February 12. Control of the illness was defined as a member with no symptoms. The dinner on February 12 was significantly associated with the illness (Mantel-Haenszel odds ratio: 3.59, 95% confidence interval: 1.06–12.20). A case-control study showed that, among the food supplied at dinner on February 12, the braised chop suey was significantly associated with the illness (odds ratio: 12.30, 95% confidence interval: 1.90–521.00). The braised chop suey had been stored in a chafing dish. An environmental investigation indicated that *Clostridium perfringens* (*C. perfringens*) in the chafing dish proliferated under an inappropriate heat-retention temperature, and the contaminated braised chop suey could have caused the food poisoning. This study demonstrated that the recommended heat-retention temperature (over 65 °C) should be confirmed thoroughly.

Key words: outbreak, *Clostridium perfringens* (*C. perfringens*), epidemiology, food poisoning

C *lostridium perfringens* (*C. perfringens*) is a commonly identified cause of foodborne outbreaks [1, 3, 5–8, 14]. A *C. perfringens* gastroenteritis outbreak usually occurs after many (more than one-hundred-thousand organisms per gram of food) organisms are ingested [7, 9, 13]. Diarrhea (loose stool, watery stool)

and abdominal cramps have been reported as the main symptoms [1–3, 5, 8, 9, 11, 14]. Vomiting and fever are usually absent [1, 3, 9, 11, 14]. Its incubation period has been reported to be approximately between 8 h and 12 h [7, 8, 10].

C. perfringens outbreaks have been reported to occur at outdoor events, especially those at which food is supplied to a large number of people [1, 5]. *C. perfringens* is ubiquitous in the environment. Therefore, food can be easily contaminated by this microorganism [1, 3,

4]. In addition, food made with meat has often been reported as a cause of outbreak due to *C. perfringens* [1, 11, 13, 14]. Therefore, the number of patients in a *C. perfringens* foodborne outbreak tends to be much larger than that in other foodborne outbreaks [8]. For these reasons, it is difficult to identify the transmission mode in a foodborne outbreak due to *C. perfringens* because there are usually many candidates.

This report describes a foodborne outbreak in which the causal food and the transmission mode were estimated, and the confirmation of the heat-retention temperature of a chafing dish could have been effective in preventing *C. perfringens* gastroenteritis.

Materials and Methods

Background. At 10:30 a.m. on February 13, 2002, a public health center in Hiroshima Prefecture, Japan, was notified that many individuals at the Japan Maritime Self-Defence Force (JMSDF) base had symptoms resembling those of food poisoning. In the JMSDF, all members were supplied the same meals (breakfast, lunch, dinner) on February 12.

Epidemiological investigation. At noon on February 13, 2002, self-administered questionnaires were distributed to all 281 members of the JMSDF. The questionnaire requested information regarding meal consumption on February 10, 11, and 12, the meal consumption time (the time the meal was eaten), symptoms, and the time of symptom onset. All 281 questionnaires were collected (collect rate = 100%). However, some questionnaires were not completed perfectly.

A case of the illness was defined as an individual who had had watery or mucousy stool, or loose stool with abdominal cramps, more than twice a day after dinner on February 12. Control of the illness was defined as an individual showing no symptoms. Controls were compared with the cases regarding the history of meal consumption. Twenty-seven members who were not included as a case or a control out of the 281 members were excluded from the analysis. The data were analyzed using Epi Info 2000.

Bacteriological investigation. Stool samples from 73 out of the 108 members with symptoms were collected and cultured for *Staphylococcus aureus*, *Salmonella*, *C. perfringens*, *Campylobacter*, *Vibrio parahaemolyticus*, and *E. coli*. Tests for *C. perfringens* enterotoxin (CPE) of these 73 stool samples were also

conducted. A bacteriological investigation regarding stool samples was conducted at noon on February 14. Furthermore, food samples on February 10, 11, and 12, and smears of the cooking area were collected and cultured for *Staphylococcus aureus*, *Salmonella*, *C. perfringens*, *Campylobacter*, *Vibrio parahaemolyticus*, and *E. coli*. A bacteriological investigation of food samples was conducted on February 13.

Environmental investigation. Food preparation workers were interviewed regarding their methods of food preparation, food preparation time, their handling methods, and the foods' ingredients. An environmental investigation was conducted at noon on February 13.

Results

Epidemiological investigation. All 281 self-administered questionnaires were collected. Eighty-one cases and 173 controls met the respective definitions among the 281 members. All of the members were male. The members' ages ranged from 18 to 53. The symptoms of the ill members were diarrhea (99%), abdominal cramps (57%), fever (5%), and nausea (3%). The epidemic curve of the 80 cases who completed information regarding the time of first symptom's onset among all 81 cases showed a common-source pattern (Fig. 1).

The number of cases and controls at each meal on February 12 is shown in Table 1. The odds ratios and their 95% confidence intervals were 1.91 (0.90–4.40) at breakfast, 4.57 (1.05–41.60) at lunch, and 5.00 (1.47–26.50) at dinner.

A stratified analysis for controlling the dinner consumption as a potential confounder showed that the lunch on February 12 was not significantly associated with the illness (Mantel-Haenszel odds ratio: 2.57, 95% confidence interval: 0.56–12.00). A stratified analysis for controlling the lunch consumption as a potential confounder showed that the dinner on February 12 was significantly associated with the illness (Mantel-Haenszel odds ratio: 3.59, 95% confidence interval: 1.06–12.20). Rice, fried food with sesame, sausage, cabbage, braised chop suey, potsticker soup, and yogurt were supplied at the dinner on February 12.

A result of the case-control study showed that the braised chop suey was significantly associated with the illness (odds ratio: 12.30, 95% confidence interval: 1.90–521.00) among the food supplied at dinner on February 12 (Table 2). The results of the stratified

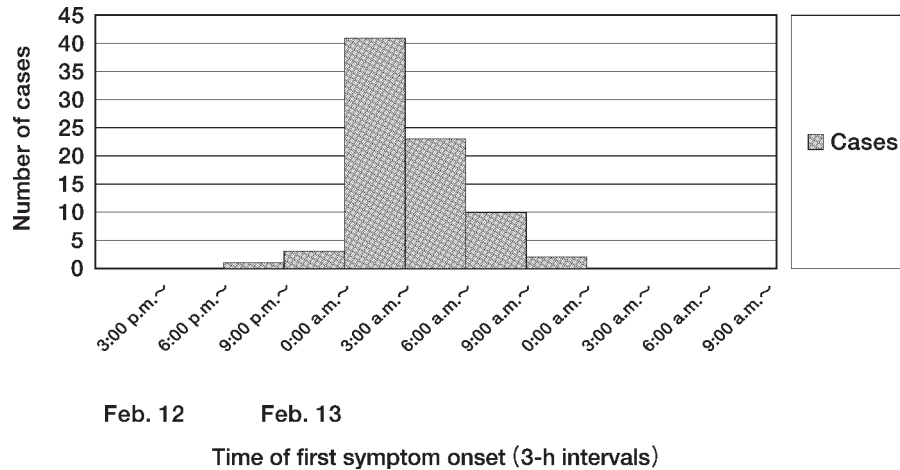


Fig. 1 Epidemic curve (time of first symptom onset of the 80 cases who completed information about the time of first symptom onset among the 81 cases, and the number of cases).

Table 1 Meal-specific odds ratios and their 95% CI* regarding illness among members who ate meals on February 12, 2002

Meal	Cases		Controls		Odds ratio	95% CI*
	Number who ate	Number who did not eat	Number who ate	Number who did not eat		
Breakfast on February 12	70	11	133	40	1.91	0.90- 4.40
Lunch on February 12	79	2	155	18	4.57	1.05-41.60
Dinner on February 12	78	3	145	28	5.00	1.47-26.50

*95% confidence interval.

Table 2 Food-specific odds ratios and their 95% CI* regarding illness among members who ate dinner on February 12, 2002

Food	Cases		Controls		Odds ratio	95% CI*
	Number who ate	Number who did not eat	Number who ate	Number who did not eat		
Rice	78	0	142	2	∞	0.10-∞
Fried food with sesame	78	0	140	5	∞	0.50-∞
Sausage	74	3	131	14	2.63	0.70-14.70
Cabbage	64	13	135	10	0.37	0.14-0.96
Macaroni salad	75	3	138	7	1.27	0.28-7.81
Braised chop suey	77	1	124	20	12.30	1.90-521.00
Potsticker soup	77	1	137	8	4.47	0.58-202.00
Yogurt	71	7	133	12	0.92	0.32-2.87

*95% confidence interval.

analyses for controlling the foods except braised chop suey as potential confounders indicated that the foods except braised chop suey were not significantly associated with the illness.

Among the cases and controls who ate the braised chop suey at the dinner on February 12, 16 cases and 90 controls completed information regarding the meal consumption time. These cases and controls were compared regarding meal consumption time. The meal consumption time of the cases was later than that of the controls (Fig. 2). The mean meal consumption time on February 12 among the cases was 5:40 p.m. (range, 5:00 p.m. to 6:05 p.m.) and that among the controls was 5:17 p.m. (range, 4:00 p.m. to 6:00 p.m.). The median time between the dinner consumption time on February 12 and illness in the 16 cases was 11.5 h (range, 10.5 h to 17.5 h). The epidemic curve of the 16 cases used in Fig. 2 was similar to the epidemic curve shown in Fig. 1.

Bacteriological investigation. *C. perfringens* was identified in 29 out of 73 stool samples from members with symptoms. CPE was identified in 29 out of 73 stool samples. *C. perfringens* was not identified in any food sample taken from February 10, 11, and 12. Similarly, *C. perfringens* was not identified in smears of the cooking area.

Environmental investigation. The ingredients of the braised chop suey were prepared in the period between 9:00 a.m. and 10:00 a.m. on February 11. The ingredients were pork, squid, Chinese cabbage, carrots, green bell peppers, and onions. After the preparation,

the ingredients were preserved in the refrigerator from 10:00 a.m. on February 11 to 3:00 p.m. on February 12. They were then cooked from 3:00 p.m. to 3:30 p.m. on February 12. The core temperature of the braised chop suey during cooking was confirmed to reach the recommended cooking temperature (over 75 °C). After the cooking, the braised chop suey was sampled for preservation, and the remainder was transferred to a chafing dish. The chafing dish had been stored on a shelf without a door under the cooking table. The braised chop suey was preserved in the chafing dish from 3:30 p.m. on February 12. At 4:00 p.m. on February 12, the braised chop suey was supplied to individuals for the self-service dinner on February 12. In the investigation, it was found that the heat-retention temperature in the chafing dish was not confirmed to reach the recommended temperature (over 65 °C).

Discussion

In the epidemiological investigation, both the main clinical symptoms in the outbreak and the mean incubation period for the food poisoning were consistent with those of *C. perfringens* food poisoning reported in previous studies [1-3, 5, 7-11].

Eighty-one individuals among the 281 met the case definition. The epidemic curve indicated that the outbreak was caused by a single exposure to a meal that was supplied before 11:00 p.m. on February 12. From the odds ratios and their 95% confidence intervals, the cause

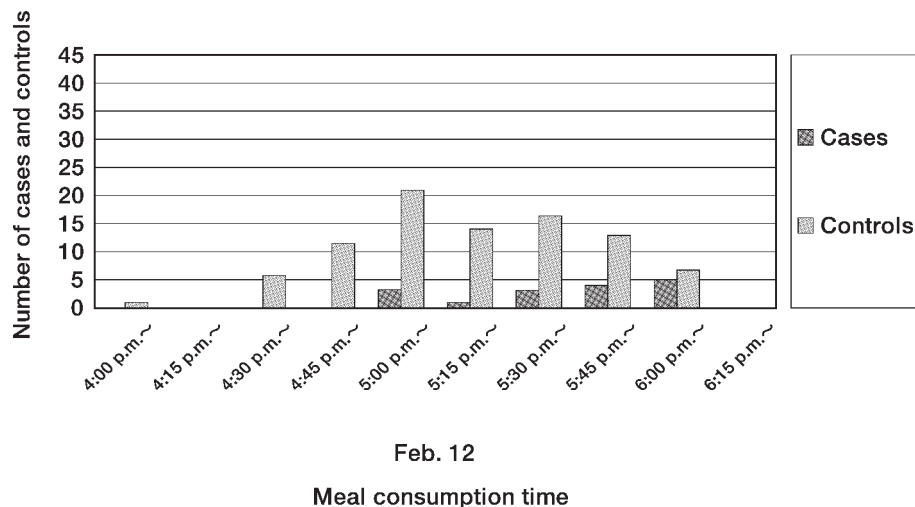


Fig. 2 Meal consumption times of the 16 cases and 90 controls who ate braised chop suey at dinner on February 12 and completed information about the meal consumption time among all 281 members in the JMSDF, and the number of cases and controls.

of this outbreak was likely to be the braised chop suey supplied at dinner on February 12. Therefore, the result of the epidemiological investigation showed that the cause of this outbreak was the *C. perfringens*-contaminated braised chop suey.

The bacteriological investigation identified both *C. perfringens* and CPE in 29 stool samples. A previous study reported that both *C. perfringens* and CPE were identified from stool samples of *C. perfringens* food poisoning patients in a *C. perfringens* foodborne outbreak because *C. perfringens* produces CPE in the human small intestine [14]. Therefore, the result of the bacteriological investigation supports the result of the epidemiological investigation.

As stated above, the results of the environmental investigation determined that the chafing dish used for the braised chop suey had been stored on a shelf without a door under the cooking table. The position of this chafing dish could have easily allowed contamination by *C. perfringens* [11]. This means that the transmission mode of the outbreak might have been the *C. perfringens*-contaminated chafing dish.

The environmental investigation also suggested that inappropriate heat retention in the chafing dish led the heat-resistant *C. perfringens* to proliferate, and that the contaminated braised chop suey caused the food poisoning. The heat-retention temperature of the chafing dish was not checked when the braised chop suey was stored in the chafing dish. The heat-retention temperature of the chafing dish could have been inappropriately low. Therefore, the chafing dish could have been maintained under optimal conditions for the growth of heat-resistant *C. perfringens*. Under these conditions, inappropriate heat retention kills heat-sensitive *C. perfringens* and other germs, and releases oxygen from braised chop suey [11]. As a result, the inside of the braised chop suey might have become an anaerobic environment. Previous studies have reported that such conditions help heat-resistant *C. perfringens*, which is likely to cause food poisoning, to proliferate [3, 9–11, 14]. Furthermore, it has been reported that *C. perfringens* proliferates twice every 8 min [3]. Therefore, the time lapse could have led the heat-resistant *C. perfringens* in the braised chop suey to proliferate, and the individuals who ate later could have been exposed to more a great number of *C. perfringens*. Fig. 2 supports the hypothesis that the time lapse allowed the heat-resistant *C. perfringens* in the braised chop suey to proliferate.

In our study, exposure and disease information were obtained from a self-administered questionnaire. Therefore, there was an information bias distorting the estimates toward the null. However, it seems that this information bias did not influence our results, because the case-control study showed that the braised chop suey was the causal food in this outbreak. In the first case-control study analyses, there were elevated odds ratios for both lunch and dinner on February 12, 2002. These increases of the odds ratios can be explained by the confounding factors, because the stratified analysis showed that the dinner on February 12 was the causal meal in this outbreak.

In the *C. perfringens* foodborne outbreak that occurred in the JMSDF, the causal food was the braised chop suey supplied at dinner on February 12. The braised chop suey was contaminated by a *C. perfringens*-contaminated chafing dish. In the chafing dish, the *C. perfringens* proliferated under inappropriate heat retention and the contaminated braised chop suey caused the food poisoning. Therefore, as time passed, members who ate the braised chop suey could be contaminated by more a great number of *C. perfringens*. Based on the results of this study, we present the following 2 recommendations. One is that containers such as chafing dishes for food should be stored under sanitary conditions. The other is that the recommended heat-retention temperature (over 65 °C) should be confirmed more thoroughly.

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References

1. Roach RL and Sienko DG: *Clostridium perfringens* outbreak associated with minestrone soup. *Am J Epidemiol* (1992) 136: 1288–1291.
2. Regan M, Syed Q and Tunstall PJ: A hospital outbreak of *Clostridium perfringens* food poisoning-implications for food hygiene review in hospitals. *J Hosp Infect* (1995) 29: 69–73.
3. Petersen LR, Mshar R, Cooper GH Jr, Bruce AR and Hadler JL: A large *Clostridium perfringens* foodborne outbreak with an unusual attack rate pattern. *Am J Epidemiol* (1988) 127: 605–611.
4. Pollock AM and Whitty PM: Outbreak of *Clostridium perfringens* food poisoning. *J Hosp Infect* (1991) 17: 179–186.
5. Tallis G, Ng S, Ferreira C, Tan A and Griffith J: A nursing home outbreak of *Clostridium perfringens* associated with pureed food. *Aust N Z J Public Health* (1999) 23: 421–423.
6. Hook D, Jalaludin B and Fitzsimmons G: *Clostridium perfringens* food-borne outbreak: an epidemiologic investigation. *Aust N Z J Public Health* (1999) 23: 421–423.

- Health (1996) 20: 119-122.
7. Shandera WX, Tacket CO and Blake PA: Food poisoning due to *Clostridium perfringens* in the United States. J Infect Dis (1983) 147: 167-170.
 8. Parikh AI, Jay MT, Kassam D, Kociemba T, Dworkis B, Bradley PD and Takata K: *Clostridium perfringens* outbreak at a juvenile detention facility linked to a thanksgiving holiday meal. West J Med (1997) 166: 417-419.
 9. Yamagishi T, Sakamoto K, Sakurai S, Konishi K, Daimon Y, Matsuda M, Gyobu Y, Kubo Y and Kodama H: A nosocomial outbreak of food poisoning caused by enterotoxigenic *Clostridium Perfringens*. Microbiol Immunol (1983) 27: 291-296.
 10. Tavis DR, Murphy RP, Jolley JW, Harmon SM, Williams C and Brumback CL: Two successive outbreaks of *Clostridium perfringens* at a state correctional institution. Am J Public Health (1985) 75: 287-288.
 11. Loewenstein MS: Epidemiology of *Clostridium perfringens* food poisoning. N Engl J Med (1972) 286: 1026-1028.
 12. Nelson KE, Ager EA, Marks JR and Emanuel I: *Clostridium perfringens* food poisoning report of an outbreak. Am J Epidemiol (1966) 83: 86-95.
 13. Chin J: Control of Communicable Disease Manual. 17th Ed, APHA, Washington DC (2000) pp 206-207.
 14. McClane BA: *Clostridium perfringens*. Food Microbiol (2001) 2: 351-372.