PHR250
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FIRST EPIDEMIOLOGICAL PROBLEM SET

DIARRHEA AT A WAKE IN ADELAIDE, AUSTRALIA

Thirteen out of 32 persons from two states who attended a lunch after a funeral in Adelaide on 2nd June 1998 became ill with diarrhea. Most had onset of illness within 3 days, but one case occurred 8 days after, and one 28 days after the lunch. The person whose illness commenced 8 days after was the sister of one of the early onset cases, and it was her boyfriend who became ill after 28 days. The duration of illness ranged from 3 to 10 days (mean=6.2 days) with reported symptoms in addition to diarrhea being abdominal pain (11), vomiting (7) and macroscopic blood in the stool (3).

Only one person had a history of recent travel outside Australia before the funeral. This had been a medically uneventful trip to the Philippines 6 weeks previous. Similarly, in the 2 months before the funeral one person had returned from southern Queensland, one from a trip to Western Australia and the Northern Territory and one from Western Australia only. Of these travelers, only the third had suffered any illness during their travel. Six people came from Melbourne to Adelaide to attend the funeral.

Most of the food for the function was purchased the day before from the refrigerated counter of a retail outlet, transported for 20 minutes in the boot of a car and then refrigerated overnight in the kitchen of the flat where the lunch was served. During that night one item (sliced ham) was removed from the fridge and some of it used. The remainder of the ham was returned to the fridge. The person who handled the ham during the night had recovered earlier that week from a diarrheal illness contracted in the Kimberleys (northwestern Australia — across the continent from Adelaide). The cause of this diarrhea had not been determined.

Pathogens of a single genus, but at least two species, were grown from the stool of five of the cases (onsets 1, 2 (n=2), 8, and 28 days after the funeral). None of the other cases provided a stool specimen.

A cohort study implicated only the sliced ham of the foods served at the funeral lunch as a possible vehicle for this outbreak. Even including a probable secondary case (8 day incubation) who did not eat ham as a primary case and counting one of the early cases (who was not completely certain that she had eaten the ham) as a non-consumer, the relative risk was 2.77 (95% confidence limits 1.05-7.27). With these conservative case definitions the attack rate for ham eaters was 8 out of 13 (62%). The more likely situation with the late onset case as a secondary case and accepting the history that the uncertain persons belief that she probably had eaten ham gave a relative risk of 5.46 (95% confidence limits 1.40-21.27).The attack rate with these definitions was 9 of 14 (64%).

The retailer from whom the ham was purchased is a large supplier which turns over

multiple legs each week. Inspection of this premises 2 weeks after the funeral by an environmental health officer of the Adelaide City Council revealed no poor food handling practices. Laboratory cultures of ham collected at that time did not yield a pathogen. If there had been a problem at or before the retail stage we would also have expected more metropolitan cases notified unrelated to the funeral.

The suspicion is that the person recently recovered from diarrhea acquired in the north west of Australia, who handled the ham the night before the funeral, contaminated it. This person also consumed the ham but did not suffer further illness. Some person-to-person transmission at the lunch was also possible. The meal was served to a large group of people in a very small flat and one person reported that the hand towel in the bathroom became sodden from hand wiping during the afternoon. The multiple-species isolates, especially in a metropolitan outbreak, are surprising but it seems unlikely that there would be multiple sources. Nevertheless the sole isolate of an alternate species in the genus came from a person who did not eat ham and who had a flu-like illness on the day of the funeral but developed prolonged (10 days) of diarrhea the day after.

1. What is the likely pathogen in this outbreak?

2. What things might have been done to prevent the outbreak?

SEVERE ILLNESS FROM RAW OYSTERS — FLORIDA, 1981-1992

From 1981 through 1992, 125 persons with similar, often oyster-associated infections, of whom 44 (35%) died, were reported to the Florida Department of Health and Rehabilitative Services (HRS). The infections generally occurred each year from March through December and peaked from May through October. Seventy-two persons (58%) had primary septicemia, 35 (28%) had wound infections, and 18 (14%) had gastroenteritis. In patients with primary septicemia, 58 infections (81%) occurred among persons with a history of raw oyster consumption during the week before onset of illness. The mean age of these persons was 60 years (range: 33-90 years; standard deviation: 12.9 years); 51 (88%) were male. Fourteen (78%) of the patients with gastroenteritis also had raw oyster- associated illness. Their mean age was 49 years (range: 19-89 years; standard deviation: 25.7 years); seven (50%) were male.

Of the 40 deaths caused by septicemia, 35 (88%) were associated with raw oyster consumption. Nine of these deaths occurred in 1992. The case-fatality rate from raw oyster-associated septicemia among patients with pre-existing liver disease was 67% (30 of 45) compared with 38% (5 of 13) among those who were not known to have liver disease.

Results of the 1988 Florida Behavioral Risk Factor Survey (BRFS) were used to estimate the proportions of the Florida population who ate raw oysters, and the proportion of the population who ate raw oysters and who believed they had liver disease (e.g., cirrhosis). BRFS and state population data indicate that approximately 3 million persons in Florida eat raw oysters; of these, 71,000 persons believe they have liver disease. Based on the number of cases reported to the Florida HRS during 1981-1992, the annual rate of illness for adults with liver disease who ate raw oysters was 72 per 1 million adults — 80 times the rate for adults without known liver disease who ate raw oysters (0.9 per 1 million). The annual rate of death from this cause for adults with liver disease who ate raw oysters was 45 per 1 million — more than 200 times greater than the rate for persons without known liver disease who ate raw oysters (0.2 per 1 million).

1. What was the most likely pathogen?

2. What options exist to limit or prevent such illnesses in the future?

GASTROENTERITIS ASSOCIATED WITH CORNED BEEF SERVED AT A ST. PATRICK'S DAY MEAL — VIRGINIA

On March 28, 1993, 115 persons attended a traditional St. Patrick's Day dinner of corned beef and cabbage, potatoes, vegetables, and ice cream. Following the dinner, 86 (76%) of 113 persons interviewed reported onset of illness characterized by diarrhea (98%), abdominal cramps (71%), and vomiting (5%). The median incubation period was 9.5 hours (range: 2-18.5 hours). Duration of illness ranged from 1 hour to 4.5 days; one person was hospitalized.

Corned beef was the only food item associated with illness; cases occurred in 85 (78%) of 109 persons who ate corned beef compared with one of four who did not (relative risk=3.1; 95% confidence interval=0.6-17.1). Cultures of stool specimens from eight symptomatic persons all yielded $\geq 10^6$ colonies of a bacterial pathogen per gram. A refrigerated sample of leftover corned beef yielded $\geq 10^5$ colonies of the same agent per gram.

The corned beef was a frozen, commercially prepared, brined product. Thirteen pieces, weighing approximately 10 pounds each, had been cooked in an oven in four batches during March 27-28. Cooked meat from the first three batches was stored in a home refrigerator; the last batch was taken directly to the event. Approximately 90 minutes before serving began, the meat was sliced and placed under heat lamps.

Following the outbreak, Virginia health officials issued a general recommendation that meat not served immediately after cooking be divided into small quantities and rapidly chilled to $\leq$40°F ($\leq$4.4°C), and that precooked foods be reheated immediately before serving to an internal temperature of $\geq$165°F ($\geq$74°C).

1. What was the most likely pathogen in this outbreak?

2. What was the most likely problem with chilling?

3. Assuming that rapid chilling had not occurred, would the reheating recommendation have prevented this outbreak?

GASTROENTERITIS IN U.S. ARMY TRAINEES — TEXAS, 1998

During August 27-September 1, 1998, 99 (12%) of 835 soldiers in one unit at a U.S. Army training center in El Paso, Texas, were hospitalized for acute gastroenteritis (AGE). Their symptoms included acute onset of vomiting, abdominal pain, diarrhea, and fever. Review of medical center admission records for AGE during the previous year indicated that fewer than five cases occurred each month.

A U.S. Army Epidemiologic Consultation Service (EPICON) team reviewed data from the inpatient records of 90 ill soldiers. AGE was defined as three or more loose stools and/or vomiting within a 24-hour period in a soldier or employee at the training center during August 26-September 1. Illness was accompanied by a minimally elevated leukocyte count, mild thrombocytopenia, and low-grade fever. The median duration of hospitalization was 24 hours (range: 12-72 hours). Stool samples collected from persons with AGE on hospital admission were negative for bacterial and parasitic pathogens.

Interviews with food handlers in the base's two dining facilities (DF1 and DF2) revealed illness in a confection baker, who had become ill in DF1 while baking crumb cake, pie, and rolls on August 26. One other DF1 employee who was not a food handler also reported self-limited gastrointestinal illness during August 27-29. No worker in DF2 reported illness.

Cultures of food specimens from the ice cream dispenser in DF1 grew nonpathogenic coliform bacteria (*Citrobacter diversus* and *Serratia liquefaciens*); however, the sample was at room temperature before culture. *Enterobacter cloacae* coliform bacteria were cultured from the soda fountain in DF2. Water samples taken from multiple sites in the training compound and from elsewhere on post were all negative for coliform contamination.

A questionnaire about food preferences, based on the previous week's menu, was administered to 86 hospitalized soldiers (84 of whom had eaten in DF1 during the 10 days before answering the questionnaire) and to 237 randomly selected soldiers from the training unit. Of the 237 nonhospitalized soldiers, 41 (17%) did not eat at DF1 during the 10 days before answering the questionnaire; 40 (17%) had illnesses that met the case definition. Thus, cases of AGE were characterized in 126 soldiers (Figure 1).

To determine the point source of the outbreak, cases with onset during August 27-28 (n=98) were analyzed separately for odds ratios (ORs) of selected exposures (Table 1). The univariate OR for illness associated with dining at DF1 during the week before the outbreak was 9.8 (95% confidence interval=2.8-40.2). Two soldiers who ate exclusively at DF2 became ill, and one ill soldier reported not eating at either facility. Food items (crumb cake, pie, cinnamon rolls, and ice cream) and soda fountain dispensers were associated with illness by univariate analysis. Using multivariate analysis, only DF1 and the carbonated beverage dispensers

remained strongly associated with illness (Table 1).

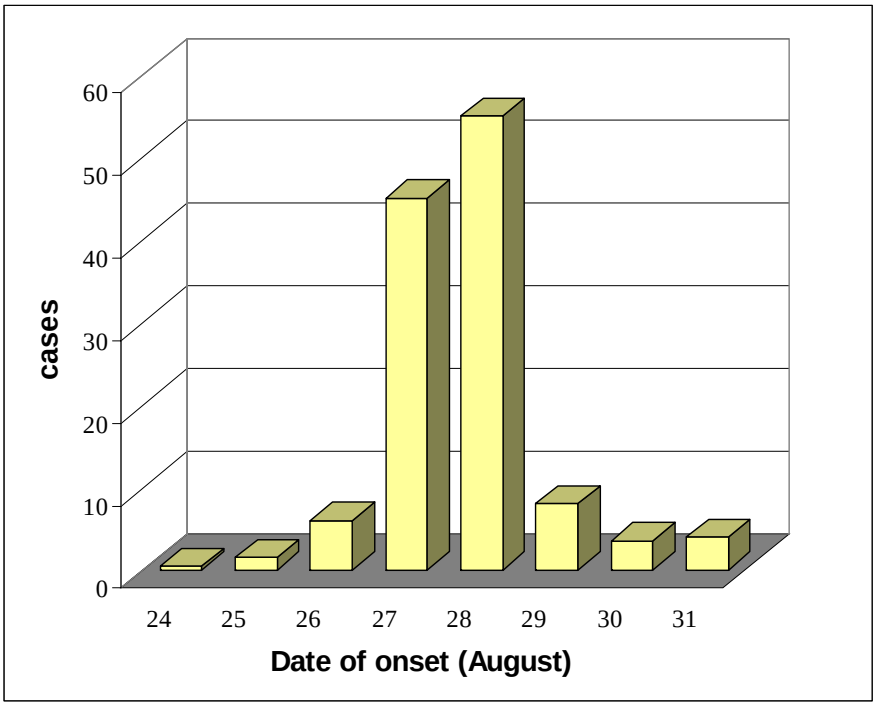

Figure 1. Number of cases of gastroenteritis among U.S. Army trainees, by date of onset — Texas, August, 1998

TABLE 1. Odds ratios for selected exposures in an outbreak of gastroenteritis in U.S. Army trainees — Texas, August 1998

	Univariate analysis		Multivariate analysis	
Exposure	Odds ratio	(95% CI*)	Odds ratio	(95% CI)
Ever ate at dining facility 1 (DF1) during the week before illness	9.8	(2.8-40.2)	7.3	(2.0-26.4)
Ate preferentially at DF1	3.7	(2.0- 6.9)	2.4	(1.3- 4.5)
Ate at dining facility 2 during the week before illness	1.1	(0.5- 2.3)	0.6	(0.2- 1.4)
Drank carbonated beverages	3.8	(2.0- 7.2)	2.6	(1.3- 5.0)
Ate crumb cake	2.4	(1.2- 4.8)	1.8	(0.8- 3.8)
Ate ice cream	1.7	(1.1- 3.0)	1.1	(0.6- 2.0)
Ate cinnamon roll	1.7	(0.8- 3.7)	1.3	(0.6- 3.0)
Ate pie	1.5	(0.9- 2.7)	1.1	(0.6- 2.0)
Used ice	1.5	(0.8- 2.9)	1.1	(0.6- 2.0)

* Confidence interval

1. What was the most likely pathogen in this outbreak?

2. What do you think the vehicle was, and how did it get contaminated?

3. If a food handler was the source of the contamination, which of them was the most likely culprit?

4. Why do reports like this tend to come more often from military establishments than from civilian populations?