Nosocomial Outbreak of Gastroenteritis Due to *Salmonella senftenberg*

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We describe a prolonged nosocomial outbreak of *Salmonella senftenberg*, an uncommon human pathogen. We detected 22 cases of infection due to *S. senftenberg* that occurred from March 1993 through November 1994 and involved 18 patients and four healthy employees. All infected persons had consumed food prepared by the hospital kitchen. The estimated attack rate for the period of the outbreak was 0.19–0.23 cases per 100,000 meals served. Infection control interventions included observation of food preparation, disinfection of kitchen devices, and education of food handlers. The consumption of lettuce (11 of 15 patients who could recount extended dietary histories vs. 4 of 20 controls; *P* = .005), cauliflower (5 of 15 vs. 0/20; *P* = .02), cottage cheese (4 of 15 vs. 0/20; *P* = .03), and deli turkey (8 of 15 vs. 0/20; *P* < .001) was associated with *S. senftenberg* infection. The isolates had identical antibiograms and pulsed-field gel electrophoretic patterns. Cultures of stool samples from food handlers as well as food items, kitchen devices, and kitchen surroundings were negative for *S. senftenberg*. Interruption of the outbreak occurred coincidentally with the institution of infection control measures. This prolonged outbreak of salmonellosis was probably related to contamination in the kitchen from turkey, with cross-contamination via equipment.

Nontyphoidal salmonellosis is an enormous public health problem in the United States; ≈40,000 cases per year were reported to the Centers for Disease Control and Prevention (CDC) over the last decade. However, an estimated 1 million to 4 million cases occur annually, with associated economic losses of $1 billion [1–26]. Most reported cases result from contamination of food from animal sources [1–6, 8, 11, 12]. Because these infections are substantially underdiagnosed and underreported, they appear sporadic in nature, but most are presumed to be part of unrecognized outbreaks [1, 8].

Health care institutions are frequent sites of recognized outbreaks of nontyphoidal salmonellosis; these outbreaks are due to the use of common kitchens, increased host susceptibility, administration of antibiotics and antimicrobials, and routine stool evaluation of diarrheal illnesses [9, 18, 19, 27–33]. From 1985 through 1991, the case-fatality rate of nontyphoidal salmonellosis was 70 times higher among patients in health care institutions than in other settings [9]. Recommendations for prevention of nosocomial outbreaks include maintaining a clean kitchen environment; cooking meat and poultry thoroughly; holding prepared food at <4°C or >60°C until it is served; avoiding cross-contamination of cooked and raw food; and following strict procedures for handling, preparing, and storing eggs [9, 19].

To detect cases of salmonellosis, hospitals with sufficient resources should perform active ward-based and laboratory-based surveillance as well as passive surveillance of employees through Occupational Health [34, 35]. When diarrheal illnesses are detected that involve employees who have had contact with patients or food handlers, stool cultures should be performed, and the employee should be removed from duty pending clinical evaluation [34, 35]. Regular education of hospital employees with respect to appropriate hygiene and infection control practices is essential [9, 19, 34, 35].

Recognition of two or more cases of gastroenteritis involving the same *Salmonella* species within a short interval should prompt a case-control investigation, and culture surveys of hospitalized patients may identify asymptomatic infected persons who can be included in the investigation [34, 35]. When hospital food is implicated as a potential source of infection, targeted microbiological sampling of the foods common to the cases may be useful, particularly when the number of cases is small, and screening of kitchen employees by means of stool cultures is recommended [34].

Despite implementation of routine infection control strategies, hospital outbreaks of nontyphoidal salmonellosis may be difficult to detect, and subsequent epidemiological investigations may prove complex. We identified a prolonged nosocomial outbreak of *Salmonella senftenberg* that occurred from March 1993 through November 1994. This report summarizes the complexities of the investigation and the control measures associated with the outbreak.

Methods

Background

Hospital A is a large midwestern facility affiliated with hospital B and a university. The physical plants of hospitals A and B are contiguous, and the two hospitals share integrated...
medical services, administrations, infection control services, laboratories, and food management services. Each hospital has a self-sufficient kitchen. Hospital A’s kitchen prepares meals daily for hospital A patients, the hospital A cafeteria, and a geographically separate university cafeteria. The infection control service at the two hospitals has 5 ½ full-time employees; these employees use a computerized, microbiology-based surveillance system. On 8 August 1994, two inpatients with *Salmonella senftenberg* infection were identified within a 4-day period at hospital A. Because of the concurrent isolation of an unusual human enteric pathogen, an investigation was begun.

**Outbreak Investigation**

*Case surveillance and presentations.* An epidemic curve of *Salmonella senftenberg* infections was created and updated as new cases were identified. A case was defined as a patient at hospital A or B, an employee of hospital A or B, or an employee of the affiliated university who had symptomatic *S. senftenberg* infection, due to any source, that was identified within the state after 1 January 1992. Secondary cases were sought through interviews with family members and co-workers of cases.

Microbiology laboratories of area hospitals, private laboratories, and local and state public health facilities were notified of the relative increase in *S. senftenberg* infections, and these laboratories were also queried regarding secondary cases. The kitchen management, department heads, nursing division heads, and three child care facilities affiliated with hospitals A and B were surveyed about reported diarrheal illnesses in the past 6 weeks; and Occupational Health records were also reviewed to identify employees with diarrheal illnesses.

On 11 August 1994, the microbiology laboratories at hospitals A and B initiated intensive surveillance for *Salmonella* species; all stool specimens submitted for detection of *Clostridium difficile* toxin were screened for *Salmonella* species as well. Retrospective reviews of the medical records of all cases were performed by the staffs of the Infection Control service and Infectious Diseases service to obtain information concerning clinical presentations, microbiology, treatment, and outcome.

*Hospital and kitchen inspections.* Inspections of one ward at hospital A (which was associated with the cases) and the kitchen at hospital A were performed. Staff members of the Infection Control and Infectious Diseases services observed and evaluated all aspects of the delivery, storage, production, preparation, and serving of food in kitchen A and provided education in food hygiene for kitchen employees. On the basis of the results of the extended dietary case-control investigation, we performed targeted cultures of food, utensils and devices, and the environment of the ward satellite kitchen and the main kitchen at hospital A. All employees who worked on the ward or in the kitchen at hospital A were evaluated for diarrheal illnesses by Occupational Health, and all hospital A kitchen employees submitted two stool specimens on separate days; these specimens were screened for *S. senftenberg*.

*Case-control study.* A data collection tool was completed with use of information obtained by review of medical records and personal interviews (when available) for all cases and all controls (selected via convenience sampling from hospital A inpatients who had been hospitalized for >14 days, after matching by date of admission). Oversampling of the controls from the hospital A ward associated with at least four cases was performed. All controls had consumed hospital A food. The information collected from these patients included demographics; occupation; medical history for the past 6 months; dates, locations, and wards of hospitalizations in the last 6 months; underlying medical conditions; risk factors for exposure to *Salmonella* species (e.g., recent travel and foods consumed); risk factors for symptomatic salmonellosis (e.g., therapy-related immunosuppression, neutropenia, prior treatment with antibiotics, or therapy with antacids and/or histamine H2 receptor antagonists); and procedures and interventions during the most recent hospitalization.

In addition, extended dietary histories were obtained through personal interviews with available cases and all controls by using a list encompassing the entire food and beverage menu of the kitchen at hospital A. The dietary histories of cases were reviewed for consumption of hospital A food in the 7 days before the onset of symptoms associated with salmonellosis. The dietary histories of controls were reviewed for consumption of hospital A food during the last 7 days of the current hospitalization.

*Microbiology.* Samples of stool, food, and environmental surroundings were selectively preenriched or enriched and inoculated onto agar, according to standard procedures [36]. Clinical samples were processed according to standard procedures, and isolates were identified by using automated techniques [36]. All *Salmonella* isolates underwent susceptibility testing with use of the disk diffusion method, somatic and flagellar antigen typing, and pulsed-field gel electrophoretic analysis of enzymatically digested genomic DNA (which was compared with that of control *S. senftenberg* isolates obtained from the California Department of Health Services) [37, 38]. Microbiology records from hospitals A and B, the State Department of Health, the CDC, and the U.S. Department of Agriculture National Veterinary Services Laboratories were reviewed for all human and animal isolates of *Salmonella* species and *S. senftenberg* recovered since 1985.

*Analysis.* Data were analyzed with use of Epi-Info Version 6.0 (CDC). Categorical variables were analyzed by means of the χ² test or Fisher’s exact test. Continuous variables were analyzed with Student’s t-test. A P value of <.05 was considered significant on two-tailed testing.

**Results**

*Outbreak Investigation*

*Case surveillance.* The two sentinel cases of *S. senftenberg* infection were identified in two hospital A patients on 8 August 1994. In a retrospective review of microbiology data, we identi-
fied 11 *S. senftenberg* infections that involved six hospital A inpatients, three hospital A employees, one outpatient (following discharge from hospital A), and one university employee; these cases (including the first case, which developed in a nurse who worked at hospital A and performed plasmapheresis) occurred between 30 March 1993 and 15 July 1994. In a prospective survey, we identified nine additional *S. senftenberg* infections that involved seven hospital A inpatients and two outpatients (following discharge from hospital A) and occurred from 12 August 1994 through 23 November 1994.

No cases were detected at hospital B, and surveillance of stool samples at this hospital was discontinued after 6 weeks. Stool surveillance cultures were continued at Hospital A through 31 January 1995, but no additional cases were identified after 23 November 1994. No further isolates of *S. senftenberg* were detected at hospital A through 1 April 1996.

The epidemic curve of the 22 identified cases revealed that an isolated case occurred in March 1993, followed by the remainder of the cases, which occurred from September 1993 through November 1994 (figure 1). No secondary infections were identified in family members or co-workers of cases. All infections involved hospital A patients (18 cases), hospital A employees (3), or university employees (1), and all cases had consumed food prepared by the kitchen of hospital A. Other identified risk factors included prior antibiotic use (11 [84.6%] of 13 cases), prior immunosuppressive medication(s) (eight [47.1%] of 17), and neutropenia (eight [36.4%] of 22).

The 15 inpatients who had *S. senftenberg* infection detected during hospitalization were located on 10 different wards; four of these patients had been housed on the bone marrow transplantation ward (six of the 15 patients had originally been admitted to the bone marrow transplantation ward, but two were transferred to other wards before the detection of *S. senftenberg* infection). No case, including the index case, had had contact with any other case.

Assuming that the kitchen was the source of the infections and based on the fact that 15,000–18,000 meals were served daily by kitchen A, the attack rate was 0.19–0.23 cases per 100,000 meals. Cases were identified in 11 of 21 months of the outbreak, and during case-months, attack rates ranged from 0.18–0.22 cases per 100,000 meals to 1.6–1.9 cases per 100,000 meals (August 1994) (*P* < .001). For the 15 inpatients, monthly attack rates by ward during case-months ranged from 1.53 cases to 5.85 cases per 1,000 patient days. The overall attack rate by ward was higher for the bone marrow transplantation ward (0.68 cases per 1,000 patient days) than for the remaining nine wards (0.11 cases per 1,000 patient days) (*P* = .008).

Case characteristics. Cases included 16 Caucasians (72.7%) and 14 females (63.6%); the median age was 36.0 years (range, 21–78 years). Eight (36.4%) of the cases were patients who had been referred to hospital A from outside the metropolitan area. One infection that occurred in the middle of the epidemic period involved a food handler who worked outside the metropolitan area. Eighteen cases (81.8%) had been inpatients at hospital A within the 6 months before the infection developed, including 15 (68.2%) who were hospitalized at the time *S. senftenberg* infection was detected. One of the 15 inpatients had been hospitalized for symptoms of salmonellosis but had consumed hospital A food multiple times before admission, and the remainder of the patients were admitted for treatment of hematologic disorders (six patients), congestive heart failure (two), or miscellaneous conditions (six). The median length of stay for hospitalized cases was 18.0 days (range, 4–127 days).

Symptoms were present in all cases, but one outpatient who developed the infection following discharge from hospital A could not be contacted for a full clinical evaluation. Symptoms among the 21 remaining cases included diarrhea in 20 (95.2%), abdominal pain in eight (38.1%), fever in six (28.6%), nausea

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**Figure 1.** The epidemic curve of a *S. senftenberg* outbreak, by quarter, from 1 January 1991 through 31 December 1995; solid portions of bars denote employee cases, and open portions denote patient cases.
in four (19.0%), vomiting in two (9.5%), and bloody diarrhea in two (9.5%) (table 1). S. senftenberg was recovered from stool for 18 (81.8%) of the 22 cases, from urine for two (9.1%), from blood for one (4.5%), and from peritoneal fluid for one (4.5%). Ten (45.5%) of 22 cases were treated for salmonellosis with antibiotics. Four of the 22 cases died, but salmonellosis did not contribute significantly to any of these fatal outcomes.

Hospital and kitchen inspections. Employees of the bone marrow transplantation ward and the kitchen of hospital A were screened by Occupational Health for recent gastrointestinal illnesses; none were detected. Inspection of the ward satellite kitchen and the main kitchen in hospital A did not reveal new kitchen procedures or hygiene problems. The staffs of the Infection Control service and the City Health Department observed the preparation, storage, and serving of food by the kitchen; no gross inconsistencies with standard infection control practices were noted.

Avoidance of the satellite kitchen and diets that included foods with a low likelihood of bacterial contamination were recommended for the patients housed on the bone marrow transplantation ward until the outbreak resolved. The utensils in kitchen A were cleaned in the steam dishwasher daily, and the devices in this kitchen were dismantled and cleaned quarterly with an iodine/sulfuric acid preparation (Mikroklene; Eco-lab, St. Paul, MN). During the outbreak, members of the Infection Control staff recommended soaking all utensils in Mikroklene overnight before steam washing, as well as complete dismantling and cleaning of devices with Mikroklene.

Case-control study. Twenty controls were selected for the study; they were similar to cases in terms of age (median age, 42.0 years; range, 17–64 years), race (68.4% were Caucasian), sex (50.0% were female), and regional home (40.0%). Controls included seven patients with hematologic disorders who were housed on the bone marrow transplantation ward and five who were in premature labor; the length of stay was longer (median stay, 28.5 days; range, 13–83 days; $P = .028$) for the controls than for cases. The proportion of controls (75.0%) who had had prior hospitalizations at hospital A during the preceding 6 months was similar to that (81.8%) of cases. Information regarding recent travel, immunosuppression, prior antibiotic therapy, and therapy with antacids and/or histamine H$_2$ receptor antagonists therapy was not consistently available for controls. Fifteen of 22 cases were available to recount extended dietary histories that spanned the length of the outbreak; of these cases, four were employees, 10 were inpatients, and one was an outpatient. Seven cases who were discharged were unavailable for this purpose; of these, five were unable to return for the interview, and two had died.

Most cases and controls were receiving a regular diet, and four cases were receiving liquid dietary supplements (none of the controls received these supplements). No cases or controls received parenteral nutrition. Hospital A had four alternating weekly menu schedules, and there was no association of cases with these schedules. Consumption of lettuce (11 of 15 cases whose dietary histories were available vs. 4 of 20 controls; $P = .005$), cauliflower (5 of 15 vs. 0 of 20; $P = .02$), cottage cheese (4 of 15 vs. 0 of 20; $P = .03$), and deli turkey (8 of 15 vs. 0 of 20; $P < .001$) was associated with S. senftenberg infection (table 2). No significant associations were noted with the remainder of the items on the menu, including 218 foods (and eight other poultry dishes) and 18 beverages served by the kitchen of Hospital A.

Hospital A purchased whole raw turkeys and whole precooked turkeys, both of which were obtained from two producers that were supplied by three poultry processors in three other states via multiple brokers. Whole raw and whole precooked turkeys were kept refrigerated side by side in their original plastic bags and cardboard boxes until use. Whole raw turkeys were roasted, and leftovers were sliced with the meat slicer and served in sandwiches to patients only. Deli turkey was prepared from whole cooked turkeys, which were sliced with the meat slicer and supplied to hospital A patients, the hospital A cafeteria, and the university cafeteria. Deli turkey was served 2–4 days each week.

Microbiology

S. senftenberg isolates from 17 of 22 patients were available for susceptibility testing. The isolates shared an identical antibiogram and were susceptible to all agents tested including ampicillin, mezlocillin, ceftizolin, cefotetan, cefazidime, imipenem, ciprofloxacin, gentamicin, tobramycin, and trimethoprim-sulfamethoxasole. All 22 isolates were identified by the State Department of Health as Salmonella group E, serovariant senftenberg. All 22 isolates shared an identical pulsed-field gel electrophoretic pattern. The patterns of three S. senftenberg isolates recovered from three cases at the beginning, middle, and end of the outbreak were distinct from the S. senftenberg isolates from controls (figure 2).

The index case was identified ~16 months after the initial infection was detected; this employee was placed on leave pending the results of stool cultures, which were performed on three consecutive days and were negative for enteric pathogens. Two stool specimens from each of 85 employees who worked in kitchen A were screened and were negative for enteric patho-

### Table 1. Signs and symptoms in 22 cases of Salmonella senftenberg infection.

<table>
<thead>
<tr>
<th>Sign or symptom</th>
<th>No. (% of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhea</td>
<td>20 (90.9)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>8 (36.4)</td>
</tr>
<tr>
<td>Fever</td>
<td>6 (27.3)</td>
</tr>
<tr>
<td>Nausea</td>
<td>4 (18.2)</td>
</tr>
<tr>
<td>Bloody diarrhea</td>
<td>2 (9.1)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>2 (9.1)</td>
</tr>
<tr>
<td>Urinary symptoms</td>
<td>2 (9.1)</td>
</tr>
<tr>
<td>Malaise</td>
<td>1 (4.5)</td>
</tr>
<tr>
<td>Chills</td>
<td>1 (4.5)</td>
</tr>
</tbody>
</table>
Table 2. Dietary histories of 20 controls and 15 cases with *S. senftenberg* infection.

<table>
<thead>
<tr>
<th>Food</th>
<th>No. (%) of cases who ate indicated food (<em>n</em> = 15)</th>
<th>No. (%) of controls who ate indicated food (<em>n</em> = 20)</th>
<th>OR (Cornfield 95% CI)</th>
<th><em>P</em> value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lettuce</td>
<td>11 (73.3)</td>
<td>4 (25.0)</td>
<td>11.00 (1.79–79.29)</td>
<td>.005</td>
</tr>
<tr>
<td>Cauliflower</td>
<td>5 (33.3)</td>
<td>0</td>
<td>NA</td>
<td>.02</td>
</tr>
<tr>
<td>Cheddar cheese</td>
<td>3 (20.0)</td>
<td>0</td>
<td>NA</td>
<td>.07</td>
</tr>
<tr>
<td>Cottage cheese</td>
<td>4 (26.7)</td>
<td>0</td>
<td>NA</td>
<td>.03</td>
</tr>
<tr>
<td>Baked chicken</td>
<td>8 (53.3)</td>
<td>4 (20.0)</td>
<td>4.57 (0.82–27.54)</td>
<td>.09</td>
</tr>
<tr>
<td>Deli turkey</td>
<td>8 (53.3)</td>
<td>0</td>
<td>NA</td>
<td>.0003</td>
</tr>
<tr>
<td>Rye bread</td>
<td>3 (20.0)</td>
<td>0</td>
<td>NA</td>
<td>.07</td>
</tr>
<tr>
<td>Salad (from salad bar)</td>
<td>3 (20.0)</td>
<td>0</td>
<td>NA</td>
<td>.07</td>
</tr>
<tr>
<td>Italian salad dressing</td>
<td>5 (33.3)</td>
<td>1 (5.0)</td>
<td>9.50 (0.83–251.77)</td>
<td>.06</td>
</tr>
</tbody>
</table>

**NOTE.** NA = not applicable (due to the presence of zero in one of four cells of the table).

* Cases from whom extended dietary histories were available.

 gens. Results of the extended dietary case-control investigation led to targeted culturing of fresh produce, raw and precooked turkey, deli turkey, and cheeses; however, all cultures were negative for *Salmonella* species. Additional cultures of suspect food items (e.g., eggs, egg-based dressings, and milk-based beverages), utensils, preparation devices (cutting boards, beverage dispensers, and slicing devices for produce, meat, and poultry), and the surroundings (tables, sinks, and drains) of the bone marrow transplantation ward satellite kitchen and the main kitchen of hospital A were negative for *Salmonella* species. During the period that the microbiology laboratory of hospital A was screening stool samples, 97 of the stool samples from patients with diarrhea that were submitted for *C. difficile* testing were screened; 10 *S. senftenberg* isolates were recovered from the stools of nine patients.

When the outbreak-related cases described in this report are excluded, the average proportion of *Salmonella* species identified as *S. senftenberg* by laboratories of the State Health Department and the CDC from 1 January 1985 through 31 December 1994 was relatively low (0.68% and 0.31%, respectively; data not shown). In contrast, the National Veterinary Services Laboratories identified 2.35% of *Salmonella* isolates as *S. senftenberg* during the same interval (data not shown).

**Discussion**

We detected an unusually large, prolonged outbreak of *S. senftenberg* infection at hospital A. Twenty-two cases were identified, but these cases likely represent <10% of all cases that occurred because of infrequent detection and poor reporting; thus, >200 persons may have developed salmonellosis during this outbreak [8]. Detection of the outbreak was delayed because the cases occurred intermittently over 21 months. In addition, the outbreak involved cases that appeared unrelated and occurred in multiple counties. The simultaneous infection of two persons with an uncommon human isolate provided impetus to initiate the investigation. However, identification of the outbreak was delayed until August 1994, by which time >50% of the cases had already occurred. Microbiology-based computer surveillance systems may be insensitive for detecting small outbreaks or outbreaks of prolonged duration; the sensi-
tivity of these systems may be increased by lowering the threshold for initiating investigations by hospitals and health departments. The use of serotype-specific rates (the number of specific serotypes/the total number of serotypes) by microbiology laboratories may prove to be more sensitive for the detection of small outbreaks.

The involvement of the patients and employees at hospital A as well as the university employee, all of whom consumed food prepared by hospital A’s kitchen, strongly implicates the kitchen as the source of the outbreak. We believe that the index case had no role in propagating the outbreak because she had no contact with any other cases, she did not have prolonged symptoms, and she did not demonstrate prolonged carriage.

Four cases were identified on the bone marrow transplantation ward, but the ward itself had no direct role in the outbreak; rather, the four cases reflect the underlying reticuloendothelial dysfunction and severe immunosuppression of the patients, resulting in greater susceptibility to symptomatic salmonellosis following ingestion of contaminated food. In addition, such patients are usually receiving broad-spectrum antibiotics, which increases the risk of developing symptomatic disease, and there is a low threshold for performing stool studies for patients on this ward because of the high prevalence of neutropenic colitis and C. difficile–related diarrhea, which likely resulted in increased case detection.

The average length of stay for cases housed on the bone marrow transplantation ward was similar to that for the other cases and controls, so the increased number of infections was not likely to be due to increased duration of exposure. These four cases, as well as the additional two cases identified following transfer, may reflect a cluster due to contamination of the ward. However, we detected no symptoms among ward personnel, culture surveys of the ward were negative, and these four cases would not explain the majority of cases that occurred during the outbreak.

The uniqueness of the isolate, the prolonged nature of the outbreak, the epidemic curve, and multiple negative cultures of specimens from the employees, the food, and environmental surfaces and devices suggest that the source of the outbreak was introduced into the kitchen of hospital A during or before March 1993 and that this initial contamination was followed by a secondary low-level contamination of a kitchen source, which continued through November 1994.

Poultry is the major recognized host of S. senftenberg [39–43], and we found consumption of deli turkey meat to be associated with infection; however, only eight of 15 interviewed cases recalled eating deli turkey. It is plausible that contaminated turkey (in a small inoculum, given the low attack rate) was the initial source of the outbreak, and that subsequent contamination of the meat slicing machine or other kitchen equipment resulted in a focus that intermittently contaminated additional foods, including other meats and/or fresh produce, via employee-mediated cross contamination. Furthermore, the hands of employees could have become transiently contaminated, resulting in transfer of the organism to other foods.

Both the intricate machine-meat interface and the presence of a biofilm complicated the cleaning of kitchen devices and could have allowed for prolonged contamination despite adequate cleaning procedures. S. senftenberg is relatively resistant to cold and heat, irradiation, and chemical agents; thus, prolonged survival of the organism may also have contributed to prolongation of the outbreak [44–55]. Interruption of the outbreak occurred coincidentally with our interventions, a circumstance suggesting that vigorous disinfection of kitchen devices (e.g., the meat slicing machine) and utensils eliminated the source and was critical in controlling the outbreak.

Ten of 22 patients received antibiotic therapy for salmonellosis, and additional undocumented therapy was possibly administered. Therapy was prescribed at the discretion of the attending physician, although the infectious diseases physicians recommended limiting treatment to immunocompromised patients with gastroenteritis, bacteremia, focal disease, and typhoid fever, according to standard practice [1, 4, 19, 24, 31, 56, 57]. Both uncomplicated gastrointestinal infection and carriage of non-typhi Salmonella (median duration, 3–4 weeks in adults) are self-limited; unnecessary antibiotic therapy exposes patients with these conditions to potential adverse reactions to medications, may prolong intestinal carriage, may result in selection for resistant organisms, and adds health care costs with minimal, if any, benefit [1, 4, 19, 24, 56, 57].

Ampicillin, chloramphenicol, and trimethoprim-sulfamethoxazole are all appropriate initial agents for salmonellosis when treatment is indicated. However, resistance, including multidrug resistance to ampicillin, trimethoprim-sulfamethoxazole, chloramphenicol, and tetracycline, occurs in S. senftenberg isolates via a large, mobile R plasmid [1, 58–60]. A third-generation cephalosporin or a quinolone may be needed in appropriate situations to eradicate infections due to resistant Salmonella species [1, 58, 55].

The outbreak reported herein has several implications for infection control. Significant nosocomial outbreaks of salmonellosis may remain unrecognized by many hospitals and health departments for substantial periods of time, as occurred at hospital A, despite maintenance of rigorous surveillance systems [34, 35]. The computerized microbiology surveillance system at this institution has proved useful for routine infection control activities, but it may be necessary to lower the threshold that triggers investigation of one case for specific pathogens (e.g., non-enteritidis salmonellosis) in order to detect clinically significant outbreaks more rapidly.

Once an outbreak is recognized, phenotypic and genotypic microbiological techniques can provide confirmation that the organisms are related [34, 35]. Despite the inherent delay during testing, pulsed-field gel electrophoresis was useful in this regard, particularly early in the investigation when demonstration of organism relatedness was crucial. Once the outbreak strain was identified and its electrophoretic pattern was determined, repeated genotypic analysis was less important given its relative rarity at this institution.

Furthermore, outbreak investigations involve time-consum-
ing observations of complex processes (e.g., food production), but these investigations may uncover deviations from standard practices and provide opportunities to reinforce good food handling practices, regardless of whether the deviations are related to the outbreak [34, 35, 61]. We noted several deficient processes that required correction: the labeling and dating of food were erratic, raw eggs were used in several dishes, eggs and produce were handled on the same counter (at different times) by the same employee, color-coded plastic cutting boards were occasionally used for the wrong type of food, and employees washed their hands inconsistently following food preparation.

On follow-up inspections, the deficiencies had been corrected; however, frequent interaction with kitchen employees, regular education regarding hygiene and control practices, and long-term follow-up will be needed to effect optimal behavior modification.

Use of the traditional pedestrian epidemiological approach provided us with numerous hypotheses regarding the cause of the outbreak, although this approach was time-consuming and the source was not identified [61]. Identification of the source of an outbreak is often difficult; in this outbreak the difficulty in identifying the source was likely due to the low level of contamination. In many outbreaks the source is not found, but the outbreak disappears; this possibly reflects improved attention to all aspects of food preparation, cleaning and decontamination procedures, personal hygiene, and routine infection control practices following identification of the outbreak [34, 35, 61].

While of unproven benefit, we recommended low-bacteria diets for patients housed on the bone marrow transplantation ward. This intervention has a sound theoretical basis, and it was simple and inexpensive to implement and should be considered for all immunosuppressed patients during a nosocomial outbreak [34]. Although it is costly, time-consuming, and unpopular with employees, routine stool screening for asymptomatic kitchen employees is generally indicated in an outbreak setting when the findings of the epidemiological investigation suggest that a kitchen source is likely [8, 10, 14, 27, 62].

Isolation of specific serotypes has provided information about the range of host and environmental niches of Salmonella species; this information may aid in outbreak investigations as well as prevention efforts [2, 3, 24]. Host ranges for three types of salmonellae are recognized: a nonspecific type (e.g., S. typhimurium), which accounts for 90% of human disease; an intermediate type with a small number of hosts (e.g., S. enteritidis); and a more specific type with few recognized hosts (e.g., S. pullorum and S. senftenberg) [2–4]. S. senftenberg is most commonly isolated from poultry—particularly turkeys—but is an uncommon human pathogen and has rarely been implicated in outbreaks of human salmonellosis [39, 40, 58–60, 63–65]. Future S. senftenberg outbreaks may be resolved more quickly with immediate attention to contaminated turkey sources.

There were several limitations to our investigation. Under-recognition of these infections (because they were asymptomatic or minimally symptomatic, because follow-up occurred outside hospital A, and because of death before detection) and underreporting of cases certainly occurred and produced sampling bias with gross underestimation of the attack rate. To minimize this bias and improve the detection of cases, we retrospectively reviewed the microbiology laboratory records at multiple hospitals as well as local and state health departments. Our surveillance activities focused attention on symptomatic hospitalized patients, so our cases (e.g., six patients with hematologic disorders) may poorly represent the affected population.

Because of the likelihood of a kitchen source, emphasis was placed on the extended dietary histories; therefore, other risk factors for salmonellosis were not thoroughly examined. However, given the involvement of both patients and employees, a kitchen source seems most plausible. Criteria for controls included prolonged hospitalization and consumption of food from the kitchen of hospital A, both of which ensured exposure to dietary sources of contamination. Nevertheless, this design was prone to sampling bias of controls. Cases and controls had different periods of exposure defined for the dietary history, which likely resulted in differential measurement bias: since a long delay from the date of infection to the date the dietary history was obtained from cases was common, poor recall would be expected to be more of a factor in the extended dietary histories of cases than of controls. Finally, we were unable to isolate the source of the outbreak and thus are unable to outline a definitive preventive strategy.

Acknowledgments

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