AN OUTBREAK OF SHIGELLA SONNEI IN BRITISH COLUMBIA – SEPTEMBER 1998

Introduction

During September 1998, four laboratory-confirmed cases of Shigella sonnei infections were reported to the British Columbia Centre for Disease Control (BCCDC) by the East Kootenay Health Services Society (EKHSS). This represented an increase over background levels for this area. All cases had eaten at a wedding dinner attended by 46 guests in Cranbrook, British Columbia, on 5 September 1998. In July and August 1998, outbreaks of S. sonnei were reported in Ontario, Alberta, Minnesota, California, Massachusetts and Florida. Uncooked parsley was epidemiologically implicated in the Minnesota and California outbreaks, and suspected in the Ontario, Alberta, and Massachusetts outbreaks.

On 22 September 1998, the BCCDC and Health Canada (Field Epidemiology Training Program) were invited by the EKHSS to assist with an investigation to identify the source of the outbreak and to determine whether it was related to the other parsley-associated outbreaks of S. sonnei reported elsewhere.

Methods

On 24 September, a retrospective cohort study was initiated among 46 persons who attended the wedding dinner catered by a local hotel. Ill persons were defined as anyone who could have consumed food or water at the wedding and who had onset of diarrhea (i.e. three or more loose stools in 24 hours) from 5 to 12 September 1998. In July and August 1998, outbreaks of S. sonnei were reported in Ontario, Alberta, Minnesota, California, Massachusetts and Florida. Uncooked parsley was epidemiologically implicated in the Minnesota and California outbreaks, and suspected in the Ontario, Alberta, and Massachusetts outbreaks.

Pathogens, Health Canada, for pulsed-field gel electrophoresis (PFGE) in relationship to the PFGE patterns of the Ontario and Minnesota isolates. A traceback investigation of parsley was conducted by the Canadian Food Inspection Agency. Data were managed and analyzed using Epi Info 6.04.

Results

Interviews were completed for 78% (36/46) of wedding guests. The remaining guests (22%) could not be reached. One person was excluded because of a diarrheal illness that began on 4 September. Thirty-four percent (12/35) of the respondents were male. The median age of the wedding guests was 43 years (range: 3 to 78).

Figure 1
Number of S. sonnei cases by date of onset (n = 13)
The attack rate for illness was 37% (13/35). Fifty-four percent (7/13) of ill persons were male. The median age of ill persons was 19 years (range: 7 to 78 years). Sixty-two percent (8/13) of ill persons were ≤ 20 years of age. The wedding dinner was self-served with adults and children going up to the buffet, and “youth” (age 13 to 24 years) going up last. “Youth” was associated with illness (RR = 2.24; 95% CI = 0.97 to 5.17; Fisher’s exact test, two-tailed P value = 0.079), although not statistically significant.

Onset of illness for wedding guests ranged from 6 to 9 September (Figure 1). The mean incubation period was 2.4 days (range: 1 to 4 days). Other than diarrhea, the most commonly reported symptoms among ill persons were abdominal cramps (84%), fever (69%), nausea (53%), and vomiting (53%). Two persons were hospitalized; no deaths were reported.

Food items associated with illness were applesauce, cocktail sauce, and punch, with only applesauce being statistically significant (Table 1). On average, ill persons ate more servings of applesauce and cocktail sauce than well persons (Table 2). The only reported food item that contained uncooked parsley (Waldorf salad) was not associated with illness. No food items contained cooked parsley.

Available food (apple juice) and water (ice water) from the wedding dinner were negative for S. sonnei. Foods collected from the same batch(28,467),(982,902) (applesauce, cocktail sauce) and same supplier (uncooked parsley) as those foods served at the wedding dinner were also negative for S. sonnei. Stool samples were collected from only four of 13 ill persons and all four were positive for S. sonnei. These isolates had the same antibiotic resistance pattern (streptomycin, sulfadiazine, tetracycline, trimethoprim-sulfamethoxazole, and ampicillin). The S. sonnei isolates were identical to one another on PFGE, but different by one band from the Ontario and Minnesota isolates.

The traceback investigation revealed that this outbreak and the event associated with the Alberta outbreak used the same Canadian importer of uncooked parsley. Parsley from this supplier was traced back to a distributor in Salinas, California.

**Discussion**

All food handlers were educated on the importance of good personal hygiene practices, and sanitary water supply and food processing. Any ill food handlers were excluded from work for the duration of their illness.

The investigation found a significant association between illness and applesauce served at the wedding dinner. However, this food could only explain two-thirds of the cases. Given the delay of up to 3 weeks between the dinner and interviews, failure of recall may account for this. An association was not found with any parsley-containing foods. The PFGE pattern from the S. sonnei isolates in this outbreak was also different from that pattern seen in Ontario and Minnesota outbreaks. Therefore, this outbreak did not fit epidemiologically with other parsley-associated outbreaks in Canada and the United States.

An alternative hypothesis is that S. sonnei was introduced by a guest to one or more foods at the wedding dinner. Given that 62% of ill persons were ≤ 20 years of age and were self-served last at the dinner buffet, it is possible that the opened applesauce, opened cocktail sauce, or both, may have been contaminated from another dinner guest who was self-served first. The median ages of ill and well persons were 19 and 45 years, respectively (Kruskal-Wallis P value = 0.146). With the exception of apple juice and ice water, no other food samples from the wedding dinner (applesauce, cocktail sauce, parsley, and punch) were available for testing.

This outbreak could not be connected by a common food to other S. sonnei outbreaks occurring at this time of the year. However, timely exchange of information on sources of outbreaks and of PFGE results will enhance the identification of bacterial contamination of foods which have a national or international distribution.

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**Table 1**

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Persons ill who ate</th>
<th>Persons ill who did not eat</th>
<th>Relative risk</th>
<th>95% CI</th>
<th>P value (Fisher’s exact test, two-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Applesauce</td>
<td>8/13</td>
<td>4/21</td>
<td>3.29</td>
<td>1.21 - 8.62</td>
<td>0.025</td>
</tr>
<tr>
<td>Cocktail sauce</td>
<td>4/5</td>
<td>9/29</td>
<td>2.58</td>
<td>1.28 - 5.18</td>
<td>0.059</td>
</tr>
<tr>
<td>Waldorf salad (containing uncooked parsley)</td>
<td>4/14</td>
<td>7/17</td>
<td>0.69</td>
<td>0.25 - 1.89</td>
<td>0.707</td>
</tr>
<tr>
<td>Punch</td>
<td>12/26</td>
<td>1/9</td>
<td>4.15</td>
<td>0.63 - 27.61</td>
<td>0.109</td>
</tr>
</tbody>
</table>

**Table 2**

<table>
<thead>
<tr>
<th>Food</th>
<th>Mean servings* eaten by persons ill</th>
<th>Mean servings eaten by persons well</th>
<th>P value (Kruskal-Wallis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Applesauce</td>
<td>0.917</td>
<td>0.273</td>
<td>0.013</td>
</tr>
<tr>
<td>Cocktail sauce</td>
<td>0.333</td>
<td>0.095</td>
<td>0.041</td>
</tr>
<tr>
<td>Punch</td>
<td>1.909</td>
<td>2.000</td>
<td>0.953</td>
</tr>
</tbody>
</table>

* Serving not quantified in questionnaire
Acknowledgements

The authors thank the following people for their assistance in the outbreak investigation: K. Ast, D. Corrigal, East Kootenay Community Health Services Society, Cranbrook BC; R. Easy, Dr. C. Clark, Dr. F. Rodgers, National Laboratory for Enteric Pathogens, Winnipeg MB; C. Sudak, Canadian Food Inspection Agency, Calgary, AB; Dr. A. Ellis, Division of Foodborne and Enteric Diseases, Laboratory Centre for Disease Control, Ottawa ON; A. Paccagnella, A. Trinidad, Provincial Laboratory, Dr. D. Skowronski, A. King, Communicable Disease Epidemiology Services, British Columbia Centre for Disease Control, Vancouver BC; Dr. E. Mintz, Diarrheal Disease Epidemiology Section, United States Centers for Disease Control and Prevention, Atlanta GA.

Source: E Nowgesic, BScN, RN, MHSc, Field Epidemiologist, J Hockin, MD, MSc, Director, Field Epidemiology Training Program, Laboratory Centre for Disease Control, Health Canada, Ottawa, ON; A Larder, MB, BCHR, Medical Health Officer, East Kootenay Community Health Services Society, Cranbrook, BC; M Fyfe, MD, MSc, Associate Director, Communicable Disease Epidemiology Services, British Columbia Centre for Disease Control Society, Vancouver, BC.

Note: An April issue of the Morbidity and Mortality Weekly Report will contain an article on outbreaks of Shigella sonnei infection associated with eating fresh parsley in Minnesota, Massachusetts, California, Florida, and Canada in July and August 1998.

Editorial Comment

A large outbreak of Shigella sonnei occurred across North America during the months of July and August 1998\(^1\). Although the only areas where this outbreak was reported were Minnesota, California, Massachusetts, Florida, Ontario, and Alberta, the widespread dissemination of the outbreak strain made it possible, perhaps likely, that the strain was imported into other areas of North America as well. The National Laboratory for Enteric Pathogens in Winnipeg found that strains with pulsed-field gel electrophoresis (PFGE) patterns identical to the S. sonnei outbreak strain were present in the greater Vancouver area during the time that the North American outbreak was at its height; however, no further outbreaks due to this organism were identified in Canada.

The Cranbrook S. sonnei outbreak described in the above report occurred later than the North American outbreak, but not so late that a connection between the two outbreaks could be definitively ruled out. Although the epidemiologic investigation of the Cranbrook outbreak implicated foods other than parsley as potential causative agents, no isolation of the outbreak strain was made from these foods. Finally, the pattern of antibiotic resistance in isolates from this outbreak was the same as was found in the North American outbreak strain.

Molecular fingerprinting investigations using PFGE confirmed that isolates from the Cranbrook outbreak had PFGE patterns differing by one band from those in the continent-wide Mexican parsley-associated outbreak. Although this suggested a high level of similarity in the PFGE patterns from both strains, it did not necessarily confirm an epidemiologic relationship. Interpretive criteria for PFGE fingerprints were developed to differentiate among strains shown to be epidemiologically related\(^2\). A difference of two to three bands would indicate that the strains were likely to be a part of the outbreak, a four to six band difference would suggest that isolates may be part of the outbreak, and a difference in seven or more bands would confirm that the isolates were different from the outbreak strain. However, as the authors of these criteria were careful to point out, such criteria cannot be safely applied when an epidemiologic connection has not been established. Barrett et al., after investigating the 1993 West Coast E. coli O157:H7 outbreak, determined that PFGE patterns of seven pre-outbreak and five sporadic isolates from the outbreak period differed in PFGE patterns from the outbreak strain by a single band\(^3\). They concluded that isolates with PFGE patterns differing by more than one band were not related, that strains differing by a single band may or may not be related, and that epidemiologic relationships cannot be determined on the basis of PFGE alone.

In a similar manner, the identification of a single band difference in the PFGE patterns of the Cranbrook and North American outbreak strains cannot be interpreted as supporting an epidemiologic relationship between the strains. The conclusion that parsley is likely not the direct causative agent of the Cranbrook outbreak-associated cases is well supported by the data. There is, however, insufficient evidence to clearly define the route of transmission of the S. sonnei strain found in this outbreak.

The North American outbreak strain was present in western Canada before the Cranbrook outbreak strain, but no are data available to show that the two strains co-existed. Similarly, unpublished data suggests that the Cranbrook outbreak strain may have been present in Vancouver at the same time as the North American outbreak strain, but there are no epidemiologic data linking the outbreaks. Given that Mexico was the source of the original North American outbreak strain, it is possible that the Cranbrook outbreak strain was imported from a similar area independently. PFGE cannot be used in this case to establish epidemiologic relationships, but it does confirm that epidemiologically related isolates from the Cranbrook outbreak were genetically identical. The source of this outbreak will probably remain an intriguing mystery.

References

This FluWatch update provides a brief summary of influenza activity up to 23 March 1999.

The weekly rates of influenza-like illness (ILI) reported to FluWatch (Figure 1) during the current season have shown a downward trend since early March, and are generally lower than those reported during the same period for the previous two seasons.

From 28 August 1998 to 19 March 1999, the FluWatch program has received reports on 40,489 laboratory tests for respiratory viruses: 4,449 (11%) were positive for influenza virus; 3,912 (88%) were influenza type A; and 537 (12%) were influenza type B. The provincial distribution of the influenza A specimens was as follows: Newfoundland (17), Nova Scotia (28), New Brunswick (57), Quebec (782), Ontario (1,506), Manitoba (179), Saskatchewan (339), Alberta (733), and British Columbia (271). The influenza type B specimens had the following provincial distribution: New Brunswick (2), Quebec (24), Ontario (473), Saskatchewan (14), Alberta (10), and British Columbia (14). These results will reflect, to some extent, local testing policy and resources.

Strain characterization has been completed on 184 influenza isolates submitted to the Bureau of Microbiology, Laboratory Centre for Disease Control, from October 1998 to 18 March 1999; 156 (85%) were A/Sydney/5/97-like (H3N2) and 28 (15%) were B/Beijing/184/93-like. The provincial distribution of the A/Sydney-like isolates was as follows: Newfoundland (3), New Brunswick (6), Quebec (35), Ontario (57), Manitoba (4), Saskatchewan (20), Alberta (23), and British Columbia (8). The provincial distribution of the B/Beijing-like isolates was as follows: Quebec (3), Ontario (18), Saskatchewan (4), and Alberta (3).

Internationally, between October 1998 and February 1999, influenza activity was reported from all of the continents. During the first 2 months of this period, influenza occurred sporadically in many countries in the northern hemisphere. Outbreaks were reported in December 1998 and, by January 1999, influenza was widespread in some countries in Africa, Asia, Europe and North America. Influenza A(H3N2) viruses predominated in some countries, influenza B viruses predominated in others, and in several countries both co-circulated. The majority of influenza A(H3N2) isolates from Africa, the Americas, Asia, and Europe were antigenically closely related to A/Sydney/5/97. Influenza B viruses from Europe and the Americas were antigenically related to B/Beijing/184/93. Influenza B viruses similar to either B/Beijing/184/93 or B/Shangdong/7/97 (a B/Beijing/243/97-like virus) continued to co-circulate in Asia (China, Japan, Singapore, and Thailand). The few laboratory-confirmed cases of influenza A(H1N1) identified and reported in Europe and North America were influenza A/Bayern/7/95-like. The majority of A(H1N1) viruses from Asia were related to A/Beijing/262/95

World Health Organization influenza reports can be accessed through the FluNet Website <http://oms.b3e.jussieu.fr/flunet>.

Reference

Erratum

MEASLES – PROGRESS TOWARD GLOBAL CONTROL AND REGIONAL ELIMINATION, 1990-1998
Vol. 25-5, Table 2, page 39

In Table 2, Measles cases and deaths in prevaccine era and in 1997, by WHO region, on page 39, under Annual cases, the numbers for Africa and for Total should read 14,477,000 (in place of 1,477,000) and 106,457,000 (in place of 104,457,000), respectively.

Source: P Buck, DVM, MSc, Field Epidemiologist, S Herman, C Scott, B Winchester, MSc, P Zabchuk, P Scockett, PhD, Chief, Division of Disease Surveillance, L Pelletier, MD, MPH, Chief, Division of Respiratory Diseases, Bureau of Infectious Diseases, M Vanderkloot, BA, Bureau of Surveillance and Field Epidemiology, LCDC, Ottawa ON; C Stansfield, MLT, Y Li, PhD, Head, Respiratory Viruses, Bureau of Microbiology, LCDC, Winnipeg MB.