Febrile Gastroenteritis Due to *Salmonella thompson*

Report of an Outbreak

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Salmonella thompson, a common pathogen of poultry, has received scant attention as a cause of human gastroenteritis. At least 45 persons were infected with S thompson *in Sacramento, California, after eating at a chicken restaurant and 38 became symptomatic. Ten required admission to hospital, and all were treated with antibiotics and improved. In 19 cases cultures of stool specimens for* S thompson *over a 60-day period showed slower but statistically insignificant differences in salmonellal elimination in 7 patients who received antibiotics when compared with 12 who were untreated. We report this outbreak to increase awareness of the virulence and prevalence of gastroenteritis due to* S thompson.

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Except for occasional case reports and notations in epidemiologic communications, *Salmonella thompson* has received scant attention as a cause of gastroenteritis.[1-5] Recently, *S thompson* caused an outbreak of gastroenteritis in Sacramento, California, in which at least 45 persons were infected and 10 required admission to hospital. In researching this species, we found that although *S thompson* is infrequently discussed in the literature, it is often listed as a cause of gastrointestinal infection and bacteremia.[6,7] We are therefore reporting this outbreak to increase awareness of the virulent gastroenteritis caused by *S thompson.*

Patients and Methods

A total of 45 persons who ate at a chicken restaurant within a four-day period were found on the basis of positive stool cultures to be infected with *S thompson.* They were interviewed within a week to determine the time of exposure and the onset, duration and type of symptoms. In all, 38 had gastrointestinal symptoms; 19 had sought medical attention and their medical records were reviewed. Ten who were admitted to hospital were studied as a separate subset to evaluate the effect of the infection on blood counts, chemistry determinations and blood culture results. Seven of the ten had stool specimens examined for fecal leukocytes and blood specimens were drawn from six to test the development of antibody to *S thompson.*

Stool specimens from 19 patients were cultured for *S thompson* over a 60-day period to determine differences in duration of excretion related to antibiotic treatment. This group comprised seven patients who were symptomatic and received antibiotics, eight patients who were symptomatic but did not receive antimicrobial therapy and four who were asymptomatic.

The source of the outbreak was investigated by the Sacramento County Health Department. This investigation included cultures of leftover foods purchased by infected persons and foods found at the restaurant.

Stool specimens were plated on MacConkey, Hektoen and VPTC agar. Lactose nonfermenting Gram-negative rods that fulfilled the biochemical and cultural criteria for *Salmonella* and that agglutinated with *Salmonella* polyvalent antiserum were further identified as *Salmonella thompson* by the California State Laboratories, Berkeley, using specific antiserum for group

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TABLE 1.—*Symptoms in 38 Patients With Gastroenteritis Due to* Salmonella thompson

Symptoms	Patients Affected	
	Number	(Percent)
Fever	21	(55)
Chills	13	(34)
Nausea	11	(29)
Vomiting	16	(42)
Abdominal cramps	20	(53)
Diarrhea	27	(71)
Headache	5	(13)

TABLE 2.—*Influence of Antibiotic Treatment on Excretion of* Salmonella thompson *in Feces*

Treatment Group	Number	Number Positive at 30 days (Percent)	Number Positive at 60 days (Percent)
Untreated	12	3 (25)	1 (8)
Antibiotics*	7	4 (57)	1 (14)

*Six patients were treated with parenteral chloramphenicol, 50 mg/kg/day for 3 days, followed by ampicillin by mouth, 50 mg/kg/day for 7 days. One patient was treated with 1 double-strength tablet of trimethoprim-sulfamethoxazole twice a day for 14 days.

C_1 antigens. The infecting strains were also tested for plasmids by Dr Mark Finch of the Enteric Disease Branch of the Centers for Disease Control, Atlanta. Minimal inhibitory concentrations of ampicillin, cefazolin sodium, chloramphenicol, tetracycline, carbenicillin, gentamicin, tobramycin sulfate, amikacin sulfate and trimethoprim-sulfamethoxazole were determined for the *S thompson* isolate by microtiter dilution techniques. Acute and convalescent antibody liters to *S thompson* were measured with a whole-cell agglutination method using a preparation containing group C antigens (Sylvania).

Results

Chicken scraps, gravy and beans collected at the homes of infected persons grew *S thompson. S thompson* was not cultured from raw chicken or other products found at the restaurant five days following the outbreak. Table 1 shows that 38 patients were clinically symptomatic with fever (21), chills (13), nausea (11), vomiting (16), abdominal cramps (20), diarrhea (27) and headache (5). The incubation period varied from 2.5 to 24 hours with a mean of 8 hours. The patients who were not admitted to hospital recovered within a few days. The ten patients requiring hospital care were more seriously ill. Their average incubation period was six hours. Their temperatures varied from 38.7°C to 40.3°C (101.6°F to 104.6°F) and they had severe gastrointestinal symptoms that persisted for 4 to 14 days. The laboratory data from these ten patients showed normal leukocyte counts of 5,300 to 11,700 per μl and large increases in band forms in the differential count. Elevations in serum enzyme activity were noted for alkaline phosphatase (two patients), aspartate aminotransferase (glutamic-oxaloacetic transaminase; two patients) and lactic dehydrogenase (one patient). Nine patients had blood cultures and one set grew *S thompson.* Four of seven stool specimens contained numerous polymorphonuclear leukocytes. In four of six patients who were tested for agglutinating antibody to *S thompson,* fourfold or greater increases in titer developed. In contrast, in none of six symptomatic patients who were not admitted to hospital did significant increases in antibody titer develop. All patients treated in hospital recovered without complications.

Table 2 contains the data for the 19 patients who had multiple stool cultures over a 60-day period. Although the differences are not statistically significant, a higher percentage of patients who received antibiotics excreted *S thompson* in their stools at 30 and 60 days after infection.

Plasmids were not found in the *S thompson* cultured from patients or from foods collected in homes and at the restaurant. The antibiograms for the *S thompson* that was recovered from the three sites were identical.

Discussion

Salmonella thompson is one of the commonest *Salmonella* serotypes infecting poultry and outbreaks are often traced to this source.[1,2,4,7,8] Investigation of the present outbreak also incriminates chicken as the probable source of infection. Chicken and gravy scraps and beans purchased from the restaurant were identified as infected foods. Because *S thompson* was not cultured from raw chicken or other products obtained at the restaurant and because employees had *S thompson* cultured from their stool specimens, it is possible that a restaurant employee who was a carrier of *S thompson* infected these products. However, because the outbreak was sharply limited and none of the employees were new, we believe that the employees, like the patrons, became contaminated and that the chicken itself was the source. Contamination of gravy and beans then permitted these foods to also serve as the vehicles for infection.

The virulence of the *S thompson* serotype is attested to by the number of patients in whom severe gastroenteritis developed. Similar virulence has been noted in previous outbreaks.[1,2] In Peru, 545 of 598 university students required admission to hospital after eating eggs contaminated with *S thompson* in the school dining room.[1] In another outbreak in Louisiana, gastroenteritis developed in 200 of 201 persons and 18 required admission to hospital. Two patients died in this outbreak.[2]

The laboratory data for the ten in-hospital patients showed that infection with *S thompson* resulted in few abnormalities. The hemograms were normal except for increases in the number of immature polymorphonuclear leukocytes. Results of hepatic and renal function tests were also normal, except for a few mild elevations in serum aspartate aminotransferase, alkaline phosphatase and lactic dehydrogenase values. *Salmonella* bacteremia occurred in only one patient, which is consistent with the reported infrequency of this event.[6] As in other studies, fecal leukocytes, which were present

in four of seven patients, were a helpful but diagnostically nonspecific finding.[9] The increases in agglutinating antibody titer in four of six patients treated in hospital, as compared with none of six outpatients, probably reflects the severity of infection. Agglutinins develop unpredictably in cases of intestinal salmonellosis and absences in antibody response are common.[10,11]

Although the differences are not statistically significant, the finding that a higher percentage of patients who received antibiotics excreted *S thompson* in their stools at 30 and 60 days after infection accords with published results showing that antimicrobial therapy prolongs the carrier state.[12,13] It is worth emphasizing that the number of carriers represents a small percentage of the infected population at two months after infection with only 2 of 19 patients having positive stool cultures. Because antibiotics prolong the carrier state and may not reduce morbidity in uncomplicated enterocolitis, many authorities discourage their use in this clinical situation.[10,12,14] These authorities recommend that antimicrobial treatment be restricted to seriously ill patients with enteric fevers, focal infections or underlying impairments in immune mechanisms.[10,14] On the basis of our observations in patients admitted to hospital with *S thompson* infection, we believe this therapeutic recommendation should be broadened to include patients who are seriously ill with gastroenteritis and in whom blood culture results are pending. Future studies may show that antimicrobial therapy is fruitless, but without this evidence it seems prudent to attempt to suppress infection in these toxic patients even if it increases the likelihood of a carrier state developing.

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