Symptomatic *Shigella sonnei* Urinary Tract Infection

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The clinical course for a patient with symptomatic urinary tract infection due to *Shigella sonnei* is described. The role of *Shigella* spp. as urinary pathogens is reviewed.

Shigella spp. usually produce self-limited gastrointestinal infections that rarely result in extraintestinal complications (2). Urinary tract infections (UTIs) due to *Shigella* spp. are rare, and *Shigella sonnei* UTIs are particularly unusual (1, 5). We report a case of symptomatic UTI due to *S. sonnei*.

Case report. A 45-year-old female presented to an outpatient medicine clinic complaining of an 8-day history of fever (temperatures taken at home peaked at 102°F [38.9°C]), chills, left-flank tenderness, polyuria, dysuria, increased frequency of urination, nausea, occasional vomiting, epigastric pain, and diarrhea consisting of five to six loose stools daily. Her past medical history consisted of a right nephrectomy, polycystic kidney disease of her remaining kidney, appendectomy, gastric bypass, dumping syndrome secondary to gastric bypass, and lumbar disectomy. When examined in the clinic, the patient had a temperature of 99°F (37.2°C), a pulse of 60/min, a blood pressure of 112/70 mm Hg, and 20 respirations per minute. The patient exhibited left-flank tenderness, but guarding and rebound tenderness were absent on abdominal examination and normal bowel sounds were noted in all quadrants. The remainder of her physical examination was consistent with her past surgical history but was otherwise unremarkable. Urinalysis revealed trace amounts of protein, 1+ bacteria, 1+ mucus threads, two to five erythrocytes per high-power field, and 25 to 50 leukocytes per high-power field; this specimen was also submitted for bacterial culture. The patient was admitted for evaluation of pyelonephritis, and intravenous therapy with ceftriaxone (1 g every 24 h) was initiated.

Significant laboratory data collected on admission are as follows: potassium, 3.1 mmol/liter; chloride, 110 mmol/liter; creatinine, 1.7 mg/dl; albumin, 3.0 g/dl; leukocyte count and differential, within normal limits. The urine specimen collected in the clinic was inoculated onto eosin-methylene blue agar and trypticase-soy agar containing 5% sheep erythrocytes (Remel, Lenexa, Kans.) by using a 0.001-ml quantitative urine loop. A gram-negative rod was recovered in quantities of >10⁵ CFU/ml on both media; it was identified as *S. sonnei* by both an API 20E strip (bioMérieux Vitek, Inc., Rockland, Mass.) and a Breakpoint Combo plate 8 (Microscan, West Sacramento, Calif.). The organism agglutinated in *Shigella* group D antiserum but failed to agglutinate in *Alkalescens*-Dispar and *Shigella* group A, B, and C antisera (Becton Dickinson Microbiology Systems, Cockeysville, Md.). As determined by breakpoint susceptibility testing (Breakpoint Combo plate 8; Microscan), the isolate was susceptible to gentamicin, tobramycin, amoxicillin-clavulanate, ticarcillin-clavulanate, cefotetan, ceftriaxone, ceftazidime, imipenem, ciprofloxacin, nitrofurantoin, norfloxacin,

tetracycline, and trimethoprim-sulfamethoxazole; it was resistant to ampicillin, piperacillin, and ampicillin-sulbactam.

On day 4 of hospitalization, a stool sample was submitted for bacterial culture and examination for ova and parasites. Stool for culture was inoculated into selenite broth and onto Hektoen, xylose-lysine-desoxycholate (XLD), *Campylobacter*-cefoperazone-vancomycin-amphotericin (CVA), and MacConkey-sorbitol agars (Remel). All agars except CVA were incubated for 48 h at 35°C in 5% CO₂ and 95% air; CVA was incubated at 42°C in a microaerophilic environment (Bio Bag type Cfj; Becton Dickinson Microbiology Systems) for 48 h. The selenite broth culture was incubated overnight at 35°C in 5% CO₂ and 95% air before being subcultured to Hektoen and XLD plates for an additional 24-h incubation at 35°C in 5% CO₂ and 95% air. The examination for ova and parasites gave negative results, and the stool culture was negative for *Salmonella*, *Shigella*, and *Campylobacter* spp. and *Escherichia coli* O157:H7. The patient had gradual resolution of all urinary symptoms but continued to produce soft stools, which were attributed to her gastrectomy-associated dumping syndrome. She was discharged after 5 days of hospitalization; antimicrobial therapy was switched at that time to amoxicillin-clavulanate (500 mg orally every 8 h) for an additional 7 days.

Discussion. Although *Shigella* spp. are highly communicable agents of bacterial diarrhea and are noted for invasion of intestinal epithelial cells, they rarely produce extraintestinal infections (2, 5). Specimens other than stool samples from which *Shigella* spp. have been recovered include those of liver, mesenteric lymph nodes, cerebrospinal fluid, synovial fluid, vaginal lesions, lungs, conjunctival sacs, corneal scrapings, blood, cutaneous lesions of the penile shaft, and urine (2, 5, 7–9).

UTI due to *S. sonnei* is extremely rare; including the current one, we are aware of only seven reported cases (1, 3, 5, 6). Of the seven reported cases of *S. sonnei* UTI, three occurred in children <12 years old, five occurred in females, and four were asymptomatic, and two patients also had positive stool cultures. The trends seen with *S. sonnei* UTIs are similar to those seen with infections by other *Shigella* spp. A review of 40 reported cases of *Shigella* UTI (including *S. sonnei* infections) indicates that 26 (65%) occurred in females, 19 (48%) occurred in children <12 years old, and all pediatric infections occurred in females (1, 3, 5, 6). Most patients (24 of 40) had symptomatic UTI, but less than half had gastrointestinal symptoms (16 of 40) or positive stool cultures (14 of 40). The most common urine isolate was *S. flexneri* (33 of 40), but most recently reported infections involve *S. sonnei*.

The route by which *Shigella* spp. gain access to the urinary tract is often unclear. It is presumed that clinical infection or asymptomatic carriage within the gastrointestinal tract provides a source for organisms that infect the urinary tract by an ascending retrograde route, particularly in females (5). Bacte-

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remia is another mechanism by which organisms might gain access to the urinary tract, but shigellemia is rare and is most likely to occur in neonates, malnourished children, and immunosuppressed (notably AIDS) patients (4, 9). Sexual transmission is also a possibility (7). The case we report is that of a 45-year-old female with symptoms of acute pyelonephritis. Urine cultures revealed *S. sonnei*, but stool cultures (which were not collected until the patient had received nearly 4 days of intravenous antimicrobial therapy) were negative. Our patient presented with gastrointestinal (along with urinary) complaints, but the significance of these complaints is confused by the symptomatology of acute pyelonephritis and her history of gastric bypass and dumping syndrome. Thus, the source of *S. sonnei* in our patient and the mechanism of spread to the urinary tract remain unknown.

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