91 Shigellosis

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Shigella is not uncommon cause of bacterial enteritis in children. It is a Gram-negative non-lactose fermenter rod. Worldwide, 90 million cases are reported annually, 89 million of which occur in developing countries. Almost 70% of cases occur in children under 5 years of age. Every year 110,000 death occurs due to shigellosis, 65% of which occur in children below 5 years of age. The main clinical presentation is that of enteritis: vomiting and diarrhea.

Microbiology

Shigella is a Gram-negative, nonmotile bacillus. There are around 36 serotypes of *Shigella* which are divided into four groups: *S. dysenteriae* (group A) including 13 serotypes, *S. flexneri* (group B) including 13 serotypes, *S. boydii* (group C) including 18 serotypes, and *S. sonnei* (group D) with one serotype. The most pathogenic type is *S. dysenteriae*, which cause a significant proportion of shigellosis in developing countries; however, it is rare in developed countries. *Shigella* can be grown on MacConkey, xylose lysine deoxycholate (XLD), and Hektoen enteric media. It is identified by its inability to ferment lactose, inability to produce H_2S, and being urease negative. Most of the cases are caused by *Shigella sonnei* (*Table 91.1*).

Pathogenesis

Shigellosis is transmitted from humans to humans by the fecal–oral route via contaminated food and water or through person-to-person contact. Few organisms 10–100 are enough to cause infections. This in addition to its ability to withstand the stomach acidity makes it easy to establish infection. After passing from the stomach, they start to multiply in the small intestine to a large number that pass to the colon where they enter the mucosal cells by induced macropinocytosis, escape from the macropinocytic vacuole, multiply and spread within the cytoplasm, and pass into adjacent cells by way of fingerlike protrusions from the cell surface. Additional pathogenic factors include elaboration of three distinct enterotoxins: members of all four species produce the virulence plasmid-encoded ShET2, strains of *S. flexneri* 2a produce the chromosomally encoded ShET1, and *S. dysenteriae* 1 produce Shiga toxin (Stx). All of these enterotoxins are able to promote the secretion of solutes and water. However, they are not the only cause of the disease due to Shigella, as non-toxigenic strains cause similar disease. *Shigella dysenteriae* 1 is able to produce a shiga toxin similar to that produced by enterohemorrhagic *Escherichia coli* O157:H7 and thus may cause hemolytic uremic syndrome.

Recently, a 110-kDa heat and trypsin-labile cytotoxin was identified and implicated to be the cause of seizure and encephalopathy that associate shigellosis.

Clinical Features

Gastrointestinal Manifestations

Shigellosis is an enteric bacterial disease that commonly manifests with diarrhea. The incubation period is usually 2–4 days ranging from 1 to 7 days. Initially, the diarrhea is watery and of large amount due to involvement of small intestine. 24–48 h later, the disease progresses to involve large intestine with the production of small frequent bowel motions that are bloody and mucoid and associated with abdominal cramps and tenesmus (bacillary dysentery). The disease is usually self-limiting and lasts for 1–2 weeks. Most of the affected children have other systemic manifestations that include fever, vomiting, and dehydration (*Table 91.2*).

Extraintestinal Manifestation of Shigellosis

Ten to 40% of patients with shigellosis may manifest seizure activity. Other CNS symptoms include headache, drowsiness, and lethargy. Bacteremia and septicemia are rare in children; however, it occurs more commonly in young infants. Vaginitis and cystitis may occur in some patients. Conjunctivitis and keratitis are also rare complications. Reactive arthritis and Rieter's disease may occur in some patients, especially in sexually active patients.

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Table 91.1
Isolated *Shigella* species from a stool culture of children at King Fahad National Guard Hospital

Shigella species	Number (%)
Shigella dysenteriae	4 (1%)
Non-speciated	22 (5%)
S. boydii	45 (9%)
S. flexneri	193 (41%)
S. sonnei	207 (44%)

Table 91.2
Clinical features due to Shigellosis

Clinical feature	Number (%)
Diarrhea	432 (92%)
Fever	393 (83%)
Vomiting	311 (66%)
Bloody stool	147 (31%)
Abdominal pain	112 (24%)
Seizure	50 (11%)

Ekiri syndrome is characterized by a fulminant course with severe dysentery, hypoperfusion, hyperpyrexia, and central nervous symptoms of convulsion and sensory impairment that progress rapidly to death. This syndrome has been described initially in Japan. Although it is rare it is being reported from other countries. Hemolytic uremic syndrome is rare and occurs mainly with *S. dysenteriae* infections and rarely with *S. flexneri* infection.

Diagnosis

The diagnostic method of choice of shigellosis is stool culture; however, stool culture is not justified in all children with diarrhea. Therefore, it should be limited to those with bloody stools or those with positive stool for leukocytes in toxic patients. Rapid test using latex agglutination test to identify *Shigella* spp. grown in the culture media is commercially available. Other diagnostic aids include leukopenia or leukocytosis with bandemia. In cases with seizures, CSF is usually normal although minority may have mild lymphoid pleocytosis. EEG is usually normal.

Treatment

Antibiotic therapy should be offered to all patients with symptomatic shigellosis. Treatment will decrease the duration of illness and also will shorten the period of organism shedding and thus will reduce secondary cases. The pattern of antimicrobial susceptibility of shigellosis is changing overtime. An increasing resistance of shigellosis to ampicillin and trimethoprim/sulfamethoxazole has been observed. Studies have shown a resistance rate of 40–80% to ampicillin and trimethoprim/sulfamethoxazole. Resistance to flouroquinolones and third-generation cephalosporins are still low in the range of 1–2%; however, regularly updated local or regional antibiotic sensitivity patterns to different species and strains of *Shigella* are required to guide empiric therapy. Empirical therapy for patient admitted with moderate or severe disease is usually by ceftriaxone until antibiotic susceptibility is available. Alternative therapy is ciprofloxacin which should not be used unless the organism is proven to be resistant to ceftriaxone. For oral therapy, cefixime and azithromycin have a good coverage in the range of 80–90%. Nalidexic acid is also a good choice but resistance is increasing.

Prevention

Hospitalized patient should be enterically isolated and hand washing should be emphasized in all contacts. No vaccine is available yet.

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