Chapter 3
Epidemiology of Dysentery Caused by *Shigella*

Epidemic dysentery in developing countries is usually caused by *Shigella dysenteriae* serotype 1 (Sd1). Sd1 is an unusually virulent enteric pathogen that causes endemic or epidemic dysentery with high death rates. It is the most common cause of large-scale, regional outbreaks of dysentery. In recent years, Sd1 has caused epidemic dysentery in Central America, south Asia and central and southern Africa. An epidemic in Central America from 1969 to 1973 was responsible for more than 500,000 cases and 20,000 deaths. The epidemic in central and southern Africa began in 1979, initially affecting eastern Zaire, Rwanda and Burundi. In the early 1990s, epidemic dysentery moved southward, affecting first Zambia, then Malawi, Mozambique, Zimbabwe and southern Africa. A large rise in the number of cases associated with refugee camps was seen in central Africa in 1994.

A. Epidemiology of *Shigella*

The genus *Shigella* is divided into four species: *S. dysenteriae, S. flexneri, S. boydii*, and *S. sonnei.* Each of these species, with the exception of *S. sonnei*, has several serotypes (Table 3-1). In general, *S. sonnei* is more common in developed countries and *S. flexneri* and *S. dysenteriae* are more frequent in developing countries. The proportions of each species vary from country to country. Sd1 differs from the other *Shigella* species in several ways:

- Only Sd1 causes large and prolonged epidemics of dysentery.
- Antimicrobial resistance develops more quickly and occurs more frequently in Sd1 than in other *Shigella* species.
- Infection with Sd1 causes more severe, more prolonged, and more frequently fatal illness than does infection with other *Shigella* species.

Table 3-1. Species and serogroups of *Shigella*

Species	Serogroup designation	Serotypes
S. dysenteriae	Serogroup A	1-13[a,b]
S. flexneri	Serogroup B	1-6
S. boydii	Serogroup C	1-18[b]
S. sonnei	Serogroup D	1

[a] *S. dysenteriae* 1 has special significance since it is unusually virulent and causes endemic or epidemic dysentery with high death rates. Monovalent antiserum (absorbed) is required to identify *S. dysenteriae* 1.

[b] Additional provisional serotypes have been reported but antisera to these new serotypes were not commercially available at the time this manual was printed.

B. Clinical Manifestations

The hallmark of infection with Sd1 is diarrhea with blood (dysentery). *Shigella* causes dysentery by invading and destroying cells that line the large intestine, leading to mucosal ulceration, a hemorrhagic inflammatory exudate and bloody diarrhea. Apart from bloody stools, patients with dysentery often have fever, abdominal cramps and rectal pain. However, the clinical response to infection spans a wide range, from mild to severe diarrhea with or without blood. In almost half of cases, *Shigella* causes acute nonbloody diarrheas that cannot be distinguished clinically from diarrhea caused by other enteric pathogens. Severity of symptoms appears to be dose related. Asymptomatic infections may occur, but not to the extent that they do in *Vibrio cholerae* O1 infections. A chronic carrier state does not occur, although the organisms may be excreted for several weeks. Sd1 infections are most often severe or fatal in young children and in the elderly and malnourished. Although most patients recover without complications within 7 days, persistent diarrhea may occasionally occur.

Infection with Sd1 can be complicated by seizures, sepsis, rectal prolapse, or toxic megacolon. A more frequent complication is the hemolytic-uremic syndrome (HUS), which is characterized by the classic triad of hemolytic anemia, thrombocytopenia and renal failure. HUS may be mild with rapid recovery, or severe, leading to kidney failure and death.

C. Treatment

The mainstay of treatment for Sd1 infection is appropriate antimicrobial therapy, which lessens the risk of serious complications and death. Other supportive measures should be used as well.

The following antimicrobial agents are currently recommended by WHO for treatment of Sd1 infections:

- ampicillin
- trimethoprim-sulfamethoxazole
- nalidixic acid
- pivmecillinam
- ciprofloxacin
- norfloxacin
- enoxacin

The selection of antimicrobial treatment should be based on recent susceptibility testing of Sd1 strains from the area or from nearby areas if Sd1 is new to the area (see Annex E). For developing a treatment policy, the antimicrobial agent chosen should be effective against at least 80% of local Sd1 strains, be given by mouth, be affordable, and be available locally or able to be obtained quickly. Unfortunately, resistance of Sd1 to ampicillin and trimethoprim-sulfamethoxazole has become widespread. Nalidixic acid, formerly used as a "backup" drug to treat resistant shigellosis, is now the drug of choice in most

areas, but resistance to it has appeared in many places. Pivmecillinam (amdinocillin pivoxil) is still effective for most strains of Sd1 but may not be readily available. Fluoroquinolones (i.e., ciprofloxacin, norfloxacin, enoxacin) should be considered only if Sd1 isolates are resistant to nalidixic acid. Fluoroquinolones are often costly and may not be readily available.

Currently, Sd1 strains are often resistant to ampicillin, trimethoprim-sulfamethoxazole, metronidazole, streptomycin, tetracycline, chloramphenicol, and sulfonamides. In addition, although Sd1 may be susceptible to some antimicrobial agents in vitro, the drug may have no documented efficacy in vivo. Examples of such agents are nitrofurans (e.g., nitrofurantoin, furazolidone), aminoglycosides (e.g., gentamicin, kanamycin), first- and second-generation cephalosporins (e.g., cephalexin, cefamandol), and amoxicillin.

Reference

World Health Organization. Guidelines for the control of epidemics due to *Shigella dysenteriae* 1. Geneva: WHO; 1995. Publication no. WHO/CDR/95.4.