Identification and management of Shigella infection in children caused by gram negative bacteria.

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Abstract

Humans are the primary reservoir of Shigella species, with captive subhuman primates as accidental hosts. In developing countries with prevailing conditions of inadequate sanitation and overcrowded housing, the infection is transmitted most often by the excreta of infected individuals via direct fecal-oral contamination. Flies may contribute to spread from feces to food. The most common species, S dysenteries and S Flexner, are also the most virulent. In developed countries, sporadic common-source outbreaks, predominantly involving S sonnet, are transmitted by uncooked food or contaminated water. The latter outbreaks usually involve semipublic water systems such as those found in camps, trailer parks, and Indian reservations. Direct fecal-oral spread can also occur in institutional environments such as child day-care centers. Mental hospitals and nursing homes. Homosexual men are also at increased risk for direct transmission of Shigella Flexner infections, and chronic, recrudescent illness complicating HIV infection has been reported.

Keywords: Tuberculosis, Anthrax, Tetanus, Leptospirosis, Pneumonia.

Introduction

Gram-negative, facultative anaerobes of the genus Shigella are the principal agents of bacillary dysentery. This disease differs from profuse watery diarrhea, as is commonly seen in choleric diarrhea or in enter toxigenic Escherichia coli diarrhea, in that the dysenteric stool is scant and contains blood, mucus, and inflammatory cells. In some individuals suffering from shigellosis, however, moderate volume diarrhea is a prodromal or the sole manifestation of the infection. Bacillary dysentery constitutes a significant proportion of acute intestinal disease in the children of developing countries, and this infection is a major contributor to stunted growth of these children. Shigellosis also presents a significant risk to travelers from developed countries when visiting in endemic areas, and sporadic food or water-borne outbreaks occur in developed countries [1].

The pathogenic mechanism of shigellosis is complex, involving a possible enterotoxin and/or cytotoxic diarrheal prodromal, cytokine-mediated inflammation of the colon, and necrosis of the colonic epithelium. The underlying physiological insult that initiates this inflammatory cascade is the invasion of Shigella into the colonic epithelium and the lamina propriety. The resulting colitis and ulceration of the mucosa result in bloody, mucous stools, and/or febrile diarrhea [2].

Possible complications of shigellosis include bacteremia, convulsions and other neurological complications, reactive arthritis, and hemolytic-uremic syndrome. Bacteremia occasionally accompanies S dysenteries serotype 1 infections in malnourished infants, but this complication is uncommon in otherwise healthy individuals. Convulsions have been reported in up to 25% of Shigella infections involving children under the age of 4 years. Both high fever and a family history of seizures are risk factors for a convulsive episode [3].

The pathogenic mechanism that underlies these pathological manifestations is diagram his cartoon incorporates experimental observations from tissue cultures and from animal models of shigellosis such as rabbit ligated ideal loops injected with virulent organisms. In the latter model, Shigella infection is initiated at the membranous cells that are associated with macroscopic lymphoid follicles. Biopsy studies in rhesus monkeys suggest that Shigella also infect microscopic lymphoid follicles of the primate colon. During the early stages of infection, bacteria are transcytosed through the M cells into the sub epithelial space. In the sub epithelial space, the organisms are phagocytized by resident macrophages. However, virulent Shigella are not killed and digested in the macrophage phagolysome [4].

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***Received:** 01-Nov-2022, Manuscript No. AAJIDMM-22-82048; **Editor assigned:** 03-Nov-2022, PreQC No. AAJIDMM-22-82048 (PQ); **Reviewed:** 17-Nov-2022, QC No AAJIDMM-22-82048; **Revised:** 21-Nov-2022, Manuscript No. AAJIDMM-22-82048; **Published:** 28-Nov-2022, DOI:10.35841/aajidmm-6.6.130*

***Citation:** Warner D. Identification and management of Shigella infection in children caused by gram negative bacteria. J Infect Dis Med Microbiol. 2022;6(6):130*

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Citation: *Warner D. Identification and management of Shigella infection in children caused by gram negative bacteria. J Infect Dis Med Microbiol. 2022;6(6):130*