



TECHNICAL BULLETIN: Pathogenic *Escherichia coli*

Although the majority of the cases of gastroenteritis are viral in etiology, bacteria often cause the disease. Bacterial gastroenteritis has many etiologies with clinical severity ranging from mild to severe. Common clinical manifestations include vomiting, diarrhea, and abdominal discomfort. Although usually self-limited, improper management of acute bacterial gastroenteritis can lead to a protracted course and potentially result in life-threatening complications. The purpose of this clinical brief is to focus on *Escherichia coli* (*E. coli*). Some *E. coli* subtypes commonly appear as colonizers of the intestinal tract within a few hours of birth but may become highly adaptive to their intestinal environment and acquire specific virulence mechanisms that can lead to a pathogenic state. Some of the genetic elements that have been acquired by these otherwise commensal microbial gut bacteria enable them to encode for disease production in normally healthy individuals. The clinical presentations of the various syndromes that are caused by these *E. coli* clones are well characterized.

- I. **Enteropathogenic *E. coli* (EPEC)** adhere to small bowel enterocytes and destroy the normal architecture of the microvillus. They induce characteristic mechanisms of attaching to the mucosal surface and produce an effacing lesion. These cytoskeletal derangements are accompanied by a host-mediated inflammatory response and clinical diarrhea, which results from multiple mechanisms that include ion secretion, increased intestinal permeability, intestinal inflammation, and loss of absorptive surface area resulting from microvillus effacement.¹

The disease that is produced is usually self-limiting and adequate fluid replacement is essential. With proper management, the prognosis for bacterial gastroenteritis caused by EPEC is very good. Mortality, although rare, is predominantly due to dehydration and secondary malnutrition from a protracted course. Antibiotics are not usually indicated unless the symptoms are severe or there is an accompanying enteric fever component.

- II. **Enterohemorrhagic *E. coli* (EHEC)** also induce the attaching and effacing lesion, but in the colon. The distinguishing feature of EHEC is the elaboration of Shiga-like toxin (*stx1* and/or *stx2*). Systemic absorption of these *stx* toxins may lead to hemolytic uremic syndrome that can potentially produce renal failure and other life-threatening complications if the patient is not properly managed. EHEC can cause both bloody and non-bloody diarrhea. One of the more recognizable strains of EHEC is *E. coli* O157:H7, but it is now recognized that only 70% of EHEC disease is caused by this serotype.²

Infants, young children, and immunocompromised patients are at risk for hemolytic uremic syndrome and hospitalization should be considered for severe EHEC infections. *Antibiotic treatment appears to increase the likelihood of developing HUS* and should only be considered if symptoms are moderate or severe. Trimethoprim-sulfamethoxazole is a firstline drug, but a parenteral second or third-generation cephalosporin may be appropriate for systemic complications.

III. Enterotoxigenic *E. coli* (ETEC) cause watery diarrhea which can range from mild, self-limiting disease to severe purging disease. The organism is an important cause of childhood diarrhea and is the *main cause of diarrhea in travelers* (particularly those who travel to developing countries). ETEC enterotoxins belong to one of two groups; the heat-labile or the heatstable enterotoxins and are structurally similar to cholera enterotoxin. This group of toxins produce activation of adenylate cyclase, increases in intracellular cAMP, the eventual activation of the main chloride channels of epithelial cells, leading to the characteristic stool consistency.²

Although often self-limiting, traveler's diarrhea requires adequate fluid replacement and the amount of fluid lost in the stools should match the amount of fluid replaced. Use of antimotility drugs can be associated with prolonging symptoms and antibiotic use should be considered in the more severe cases.

IV. Enteroinvasive *E. coli* (EIEC) are biochemically, genetically, and pathogenically related to *Shigella* sp. (they are taxonomically indistinguishable at the species level and, for that reason, are reported together). The early phase of EIEC/*Shigella* pathogenesis comprises epithelial penetration followed by lysis of the endocytic vacuole, intracellular multiplication, directional movement through the cytoplasm and extension into adjacent epithelial cells. Both organisms induce apoptosis in infected macrophages. Sloughing of the intestinal mucosa and bloody diarrhea is produced.

EIEC/*Shigella* infection produces a self-limited diarrheal illness that lasts 5-7 days and may not require antibiotics in individuals who are otherwise healthy. Antibiotic treatment is recommended for infirm or older patients, malnourished children, patients infected with HIV, food handlers, health care workers, and children in day care centers.³ For public health reasons, most experts recommend treating any person whose stool is positive for *Shigella* species; a similar rationale applies to infections caused by EIEC. Moreover, antibiotics have been shown to decrease the duration of fever and diarrhea by about 2 days. The shorter duration of shedding with antibiotic therapy can reduce the risk of person-to-person spread.



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