Bacterial gastroenteritis

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Abstract

Infectious diarrhoea is a major public health concern worldwide. Bacteria, the focus of this review, are responsible for 20–40% of diarrhoeal episodes, contributing to high rates of childhood mortality in developing regions, and substantial morbidity and economic losses in developed regions. The epidemiology is changing, with salmonellosis decreasing in industrialized countries and diarrhoeagenic *Escherichia coli* contributing to an increasing burden of disease worldwide. Molecular diagnostics has improved our understanding of the epidemiology, aetiology and pathogenesis of bacterial gastroenteritis, and revealed new pathogenic agents, although widespread introduction of such diagnostics into clinical practice will require careful validation and cost–benefit analyses. The development of antimicrobial resistance in gastrointestinal pathogens has implications for treatment options. We review the epidemiology, common aetiological agents and their clinical features, and the diagnosis, treatment and prevention of bacterial gastroenteritis, and propose an investigation and management algorithm.

Keywords *Campylobacter*; diagnosis; diarrhoea; diarrhoeagenic *Escherichia coli*; gastroenteritis; management; MRCP; pathogenesis; *Salmonella*; *Shigella*; *Vibrio cholerae*

Key points

- Most episodes of diarrhoea are self-limiting, but the risk of severe disease or complications is greater at extremes of age and in the presence of co-morbidities, such as diabetes mellitus or bowel disease
- The mainstay of therapy is rehydration with oral rehydration therapy, but antibiotics are indicated in inflammatory diarrhoea and individuals at risk of complications and can be considered in travellers' diarrhoea
- Antibiotics should be avoided in children and where Shiga Toxin associated haemolytic–uraemic syndrome is suspected
- Fluoroquinolone resistance has increased dramatically in many pathogens, particularly *Campylobacter,* and particularly in South-East Asia: as a result, azithromycin is the empirical therapy of choice
- Wider public health issues, such as infection control and notification requirements, must be considered when managing infectious diarrhoea. The implications of antimicrobial resistance should be considered where foreign travel increases the risk of imported pathogens
- Molecular advances have provided new data on epidemiology of gastrointestinal pathogens, and offer promising targets for rapid diagnosis

Introduction

Gastroenteritis refers to syndromes of diarrhoea or vomiting resulting from non-inflammatory infection in the upper small bowel or inflammatory infection in the colon. It can be caused by bacteria, viruses or parasites, but in many cases no pathogen is identified.

Bacteria, the focus of this review, are responsible for around 20–40% of diarrhoeal episodes in which an infective agent is identified in the UK;[1] however, they are likely to contribute more significantly in developing regions, where there is a greater burden of diarrhoeal disease, and associated childhood mortality.[2] Although mortality has declined with the introduction of oral rehydration therapy (ORT), morbidity is unchanged, and prolonged or recurrent diarrhoea is associated with malnutrition and adverse effects on growth and development.[2] With increasing international travel and globalization of the food industry, clinicians must be alert for imported and unusual pathogens and outbreaks, and increasing antibiotic resistance must also be considered when choosing empirical treatment.

Here, we review the changing epidemiology of bacterial gastroenteritis, its aetiological agents and developments in diagnosis, treatment and prevention, and propose an investigation and management algorithm.

Epidemiology

Infectious diarrhoea is a major public health concern worldwide. In developing countries, children <5 years old suffer 3–4 diarrhoeal episodes per year, with significant but declining mortality. In contrast, <0.3 episodes occur per person per year in developed regions, but this still equates to >5 million cases each year in the USA, 80% of which are food-borne. The vast majority do not present to healthcare facilities, but they result in substantial morbidity and economic losses estimated at billions of dollars annually.

Epidemiological aspects of each causative organism are discussed in more detail below, where relevant.

Causative bacterial pathogens

A recent UK study combining culture and molecular methods to detect intestinal pathogens found *Campylobacter* to be the most common cause of bacterial gastroenteritis,[1] particularly in more severe cases presenting to general practitioners. This was followed by enteroaggregative *Escherichia coli* (EaggEC) non-O157

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Shiga-toxin-producing *E. coli* (STEC) and non-typhi salmonellae (NTS). Screening for diarrhoeagenic *E. coli* (DEC) strains other than O157:H7 is not routinely done, however, and these appear to be increasingly contributing to diarrhoeal disease.[1,3] Over the past 20 years, food-borne outbreaks of diarrhoea have increasingly been attributed to *Campylobacter* and STEC, whereas *Salmonella* has been declining.[4]

Bacteria are also the main cause of travellers' diarrhoea (TD). Using conventional culture methods, *Shigella* and NTS are identified in most TD acquired in Africa, Latin America and the Caribbean; *Shigella*, NTS and *Campylobacter* are identified in similar proportions of TD in South Asia.[4] However, molecular techniques have shown that enterotoxigenic *E. coli* (ETEC) causes most cases in all regions, except for South-East Asia, where *Campylobacter* accounts for around 33%.[4]

Risk factors

Many host and environmental factors influence the development of bacterial gastroenteritis, including:

- **Weaning** – this leads to loss of mucosal immunity from maternal antibody.
- **Age** – young children lack immunity to certain pathogens, for example enteropathogenic *E. coli* (EPEC). Elderly individuals are at increased risk of infection caused by age-related alterations in mucus production, gut flora, gut motility and cell surface receptors for microbial adhesions or toxins, for example *Clostridium difficile*.
- **Gastric acidity** – achlorhydria, gastrectomy and use of antacids/proton pump inhibitors decrease the bactericidal effect of gastric acid.
- **Intestinal dysmotility** – this adversely affects the distribution of normal intestinal flora and prevents removal of pathogens.
- **Antibiotics** – these reduce normal intestinal flora (particularly anaerobes), thereby increasing colonization opportunities for pathogens.
- **Immunosuppression** – impaired adaptive immunity, for example HIV infection, predisposes to some enteric pathogens such as NTS.
- **Genetic predisposition** – blood group O is associated with increased susceptibility to cholera.
- **Overcrowded living conditions** – these enhance spread of organisms.
- **Poor sanitation** – this increases the acquisition of food- and waterborne infection, as well as person-to-person transmission.

Pathogenic mechanisms and associated clinical syndromes

Bacteria cause gastroenteritis by three main mechanisms, associated with distinctive but overlapping clinical syndromes (Table 1):

- Production of preformed toxin induces vomiting and abdominal cramps within a few hours of ingestion.
- Secretion of toxin after adhering to intestinal epithelium causes a syndrome of watery diarrhoea, without blood or mucus or associated fever (non-inflammatory).
- Invasion of intestinal mucosa causes dysentery, the passage of small-volume stools containing blood, mucus and pus associated with fever, lower abdominal pain and tenesmus (inflammatory).

Major bacterial causes of gastroenteritis and the most commonly associated clinical syndromes

Intoxication
- *Staphylococcus aureus*
- *Bacillus cereus*
- *Clostridium perfringens*

Watery diarrhoea
- *Vibrio cholerae*
- Non-typhi salmonellae
- Enterotoxigenic *Escherichia coli*
- Enteroaggregative *E. coli*
- Enteropathogenic *E. coli*
- Enteroinvasive *E. coli*
- *Campylobacter* spp.
- *Yersinia* spp.

Dysentery
- *Shigella* spp.
- Enteroinvasive *E. coli*
- Enterohaemorrhagic/Shiga-toxin-producing *E. coli*
- *Yersinia* spp.

Table 1

Principal bacterial causes of gastroenteritis

Campylobacter

Campylobacter jejuni and *Campylobacter coli* are leading causes of bacterial gastroenteritis worldwide, with children and young adults most susceptible. Organisms usually reside in the intestine of cattle, poultry, domestic pets and birds. Infection occurs after ingestion of contaminated undercooked food, or from close contact with infected animals. Recent UK outbreaks have been associated with chicken liver pâté and other prepared chicken products. Person-to-person transmission is uncommon. The infective dose is approximately 10^2–10^5 organisms.

Clinical presentation varies, from an asymptomatic carrier state to mild enteritis with abdominal cramps and watery diarrhoea, to dysentery with severe abdominal pain, fever and bloody diarrhoea. The incubation period is typically 1–6 days, often with a prodrome of fatigue and myalgia. Symptoms last a few days, but persist beyond a week in around 10% of cases. Complications include bacteraemia and osteomyelitis.

There is an increasing appreciation of the role of *C. jejuni* in uncommon but significant post-infectious sequelae, such as reactive arthritis, transverse myelitis, Guillain–Barré syndrome and myocarditis. *Campylobacter* has also been linked to a rare lymphoma called immunoproliferative small intestinal disease, which can respond to antimicrobial therapy in the early stages.

Symptoms are usually self-limiting. If treatment is considered (Figure 1), azithromycin 500 mg daily for 3 days is recommended[3] because of increasing fluoroquinolone resistance worldwide, particularly in South-East Asia, where rates of 80% are reported.

Diarrhoeagenic *E. coli*

E. coli is part of the normal gut flora, but at least five groups of pathogenic strains exist that cause a range of gastrointestinal

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