Intra-abdominal infections

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Gastroenteritis

Gastroenteritis can be defined as a syndrome of diarrhoea and/or vomiting after ingestion of an infectious agent or toxin. It is a major cause of morbidity and mortality worldwide, especially in children in developing countries. Infective diarrhoea is common in developed countries but mortality is low. Approximately 71,000 cases were notified to the Health Protection Agency in England and Wales in 2003. Causative agents are listed in Table 1.

Hospital admission for gastroenteritis is indicated for those with severe symptoms, and is more likely in those with diabetes mellitus, renal disease, heart disease, ileostomy or immunosuppression, or the frail elderly. It is important to remember that diarrhoea and vomiting are non-specific symptoms which may be caused by other illness including pneumonia, urinary tract infection, surgical abdominal conditions or constipation with overflow.

Features in the history from patients with suspected gastroenteritis may help with diagnosis and identification of likely pathogens (Table 2). Food history may be unreliable because of poor recall and misjudgement of the incubation period leading to the most recent meal being blamed (only helpful in preformed toxin food poisoning) rather than that consumed 2–3 days previously (important for the commonest organisms, salmonella and campylobacter). Patients will often discount meals shared with unaffected others but they may have handled the raw ingredients, be more susceptible (eg reduced gastric acidity due to proton-pump inhibitor therapy) or the organisms may have been unevenly distributed in the food.

Closure of wards due to nosocomial spread of viral gastroenteritis is common, made worse by frequent moves of patients through different wards and the lack of side rooms. Norovirus frequently causes more vomiting than diarrhoea and the diagnosis needs to be considered in all patients, especially the elderly with no other apparent cause of vomiting. Large numbers of virus particles in the vomit allow its ready transmission to other patients and staff.

Key Points

Identification of risk factors may help in the diagnosis of gastroenteritis

Closure of wards due to nosocomial viral gastroenteritis is an increasingly common problem and could be reduced by use of isolation and good hand hygiene in suspected cases

Antimicrobial therapy in gastroenteritis should be restricted to those with severe, prolonged or invasive infections or for certain specific infections

Diarrhoea and vomiting are non-specific symptoms which can be due to many causes other than gastroenteritis

Ultrasound or computer tomographic guided drainage of intra-abdominal abscesses with appropriate antimicrobial therapy may reduce the need for surgical intervention

KEY WORDS: abdominal tuberculosis, amoebiasis, antimicrobials, food poisoning, gastroenteritis, intra-abdominal abscess, liver abscess, peritonitis

Types of diarrhoea

- *Large-volume diarrhoeal illness* is usually of small bowel origin with loss of electrolytes and fluids. Common pathogens are noroviruses, rotavirus, salmonellae, toxin-producing *Escherichia coli*, preformed toxin food poisoning, giardia and cryptosporidiosis.
- *Small-volume diarrhoea with blood and mucus* is usually of large bowel origin and is characteristic of invasive pathogens such as campylobacter, shigella, enteroinvasive *E. coli*, and amoebiasis. Verotoxigenic *E. coli* (VTEC) (eg type O157) and *Clostridium difficile* also cause large bowel diarrhoea due to elaboration of cytotoxins.
- *Watery diarrhoea becoming bloody* is seen in campylobacter and VTEC infections.

If the symptoms are prolonged (more than three weeks), giardiasis, cryptosporidiosis (usually in the immunocompromised) or a non-infective cause should be considered as other microbial causes would usually have resolved by then.

Examination and investigations

Dehydration may cause tachycardia, hypotension, dry mucous membranes or decreased skin turgor. Abdominal tenderness is usually present; it can be severe such that surgical diagnoses may need to be considered.

Light microscopy of faeces (with appropriate staining) will identify cryptosporidiosis, giardiasis and *Entamoeba histolytica* as well as other less common parasites. Culture on selected media, with subsequent sensitivity tests as appropriate, will identify most bacterial pathogens. Toxin assay is used to detect *C. difficile* as culture does not distinguish between colonisation and active disease. Viral studies such as enzyme immunoassay (EIA) for rotavirus, adenovirus and norovirus should be requested in outbreaks or on clinical suspicion of viral gastroenteritis in hospital

Table 1. Causes of gastroenteritis.

Toxin (preformed)	Bacterial	Viral	Protozoal
Staphylococcus aureus	**Campylobacter**	**Rotavirus**	**Giardia**
Bacillus spp	**Salmonella**	**Norovirus**	**Cryptosporidium**
Clostridium perfringens	**Shigella**	(eg **Norwalk and**	*Entamoeba histolytica*
Diarrhoeal shellfish poisoning	***Clostridium difficile***	**other SRSVs**)	Cyclospora
(due to toxic algae)	***Escherichia coli:***	Adenovirus type 40/41	Isospora
	verotoxigenic enterotoxigenic	Astrovirus	
	enteroinvasive enteropathogenic		
	Vibrio cholerae & other vibrios		
	Clostridium perfringens		
	Yersinia		
	Aeromonas		

Bold type indicates commoner agents in the UK.
SRSV = small round structured virus.

inpatients. Electron microscopy is labour intensive and costly so should be restricted to investigation of outbreaks where EIAs are negative. Duodenal sampling may be necessary to diagnose giardiasis as faecal microscopy may fail to detect the sparse number of cysts. Serology for antibodies to *E. histolytica* in invasive amoebiasis or yersiniosis may be helpful.

Blood cultures should be done if there is fever, but also in severe illness and in the immunocompromised even if apyrexial. Routine abdominal radiographs should not be performed in investigation of gastroenteritis, but can be helpful to confirm faecal loading in patients who may have overflow diarrhoea or possible intestinal obstruction, or to check for toxic dilatation in severely ill patients.

Complications

The commonest acute complication is renal failure, usually prerenal due to dehydration, but this may progress to established tubular necrosis. Haemolytic-uraemic syndrome (HUS) is more common in children and most often associated with VTEC (*E. coli* O157) but is also seen occasionally with shigella (usually *Shigella dysenteriae*) or salmonella infection. Septicaemia is most commonly due to salmonellae, where the patient may go on to develop more indolent symptoms similar to typhoid or develop metastatic infection (eg splenic abscess or osteomyelitis).

Toxic megacolon is a rare occurrence, usually in invasive dysenteric illness. Amoebic disease is classically associated with liver abscess (see below), although this can occur without preceding dysentery.

Postinfective complications include temporary lactose intolerance or malabsorption (more common in infants), irritable bowel syndrome,[1] erythema nodosum, reactive arthritis and Guillain-Barré syndrome.

Management

The mainstay of treatment involves rehydration, which can be oral or intravenous depending on severity and ability

Table 2. Examples of risk factors for gastroenteritis.

Risk	Common pathogen(s)
Low gastric acidity – proton pump inhibitor therapy, achlorhydria	Salmonella, campylobacter
Exposure to farm animals	Cryptosporidium, *Escherichia coli* O157
Recent or current antibiotic therapy	*Clostridium difficile*
Hospital acquired:	
patient	Norovirus, *Clostridium difficile*
staff	Norovirus
Immunodeficiency	Salmonella, giardia, cryptosporidium, isospora
Drinking water purified by chlorination only*	Cryptosporidium
Travel:	
tourist resorts abroad	Giardiasis, salmonella, campylobacter
developing countries	Giardiasis, shigellosis, amoebiasis
Contact with others with diarrhoea	Viral, shigella
Drinking unpasteurised milk	Salmonella, campylobacter, cryptosporidium
Eating raw eggs	Salmonella
Eating rare beefburgers†	*Escherichia coli* O157
Poorly stored cooked meat dishes	*Clostridium perfringens*
Domestic pets with diarrhoea	Campylobacter
Contact with reptiles	Salmonella

* Cryptosporidium cysts are removed by filtration but are resistant to chlorination and may occasionally contaminate the drinking water supply. Severely immunosuppressed patients should drink boiled water.
† *E. coli* O157 originally on the surface of meat may reach the centre of burgers after mincing where bacteria are less susceptible to cooking.

to tolerate oral fluids. Diuretics and other antihypertensive medication worsen dehydration so may need to be temporarily withheld. Symptomatic treatment with antispasmodics (eg dicycloverine, simple analgesics and antiemetics) often helps. Antidiarrhoeal agents (eg loperamide) may help mild to moderate cases.[2] They are usually ineffective in severe (hospitalised) cases and can precipitate toxic megacolon in severe invasive disease, so are best avoided in inpatients. Patients should be nursed in single rooms with *en suite* toilet or dedicated commode unless they are part of a hospital outbreak in which case patients may be grouped together. Universal infection control precautions must be applied and the importance of hand hygiene should also be emphasised to patients and their visitors.

Antimicrobial therapy is usually unnecessary, but should be considered in severe or prolonged illness, the immunosuppressed, the elderly, those with comorbidity (eg ischaemic heart disease) and when blood cultures are positive. Specific infections may warrant antimicrobial treatment (Table 3). Antimicrobials have no effect on uncomplicated salmonella infection and may prolong carriage[3] and possibly increase the severity of HUS, although this is not supported by meta-analysis.[4]

It is reasonable to try empiric treatment with metronidazole in suspected giardiasis to prevent the need for endoscopy but metronidazole resistance,[5] which in our experience is increasing,

Table 3. Treatment of bacterial gastroenteritis.

Cause	Treatment	Notes
Campylobacter	Erythromycin, ciprofloxacin	Only in severe illness or persistent symptoms Ciprofloxacin resistance in about 15%
Salmonella	Ciprofloxacin	Only in invasive disease
Shigella	Ciprofloxacin	Antimicrobials not used for milder illness due to *Shigella sonnei*
Clostridium difficile:		
acute	Metronidazole, oral vancomycin	Mild cases: withdrawal of triggering antimicrobial may be sufficient Vancomycin reserved for metronidazole failure or second relapse
recurrent	Prolonged/tapered metronidazole or vancomycin[6]	*Saccharomyces boulardii* may help,[7] cholestyramine has been used
Escherichia coli O157	Supportive	Antimicrobials may increase HUS (see text)
Giardia	Metronidazole, tinidazole	Clinically resistant disease: albendazole, mepacrine
Cryptosporidium	Supportive	Paromomycin may help in immunocompromised
Entamoeba histolytica	Metronidazole	Diloxanide or paromomycin to eradicate luminal cysts
Empiric treatment in severe illness or immunocompromised	Ciprofloxacin and metronidazole	

HUS = haemolytic-uraemia syndrome.

Table 4. Empiric antimicrobials in intra-abdominal sepsis.

	Common pathogens	Examples of therapy for moderate illness	Less common pathogens	Examples of therapy for life-threatening illness
Peritonitis:				
secondary to perforation	Enterobacteriaceae, anaerobes	Cefuroxime, metronidazole	*Pseudomonas aeruginosa*, enterococci	Imipenem or piperacillin/tazobactam
primary	Enterobacteriaceae, pneumococcus	Cefotaxime	Enterococci	Imipenem or piperacillin/tazobactam
dialysis related	Enterobacteriaceae, *Staphylococcus aureus*	ip vancomycin and oral quinolone	*Pseudomonas aeruginosa*, *Staphylococcus epidermidis*	iv vancomycin and ceftazidime
Intra-abdominal abscess	Enterobacteriaceae, *Streptococcus milleri*, anaerobes	Cefuroxime, metronidazole	Enterococci	Piperacillin/tazobactam or imipenem

ip = intraperitoneal; iv = intravenous.

means that a poor response does not exclude giardiasis.

Prevention

Prevention relies on food and water safety and good hygiene practices, especially hand washing. Investigation of outbreaks and food poisoning is the responsibility of environmental health and the consultant in communicable disease control. It is a statutory requirement to notify a case of gastroenteritis. Early recognition and subsequent isolation of patients with gastroenteritis is a key factor in the prevention of hospital outbreaks.

Peritonitis

Peritonitis most commonly arises after perforation of a viscus although it may be secondary to rupture of an abscess. Such patients are extremely unwell, with severe abdominal pain and rigidity, vomiting and shock. They require supportive management, including empiric antibiotics (Table 4) and usually surgery. Peritoneal infection due to a dialysis catheter usually causes abdominal pain, fever and cloudy dialysis fluid. It can be managed by antibiotics, initially given via the catheter (Table 4). Primary peritonitis may occur in patients with ascites due to cirrhosis or the nephrotic syndrome. Blood cultures and peritoneal fluid culture should be taken before starting antibiotic therapy. Coliforms and pneumococci are most commonly isolated.

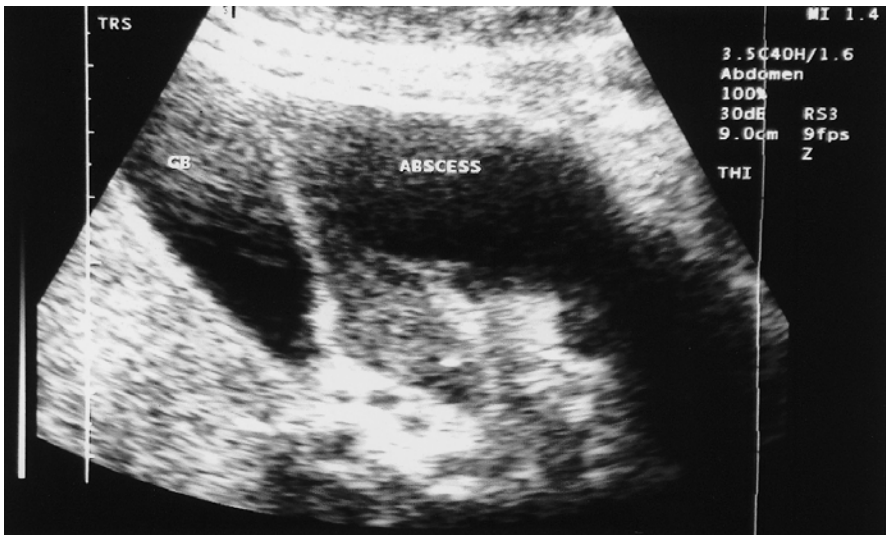

Fig 1. Liver ultrasonography showing large echopoor area due to a liver abscess.

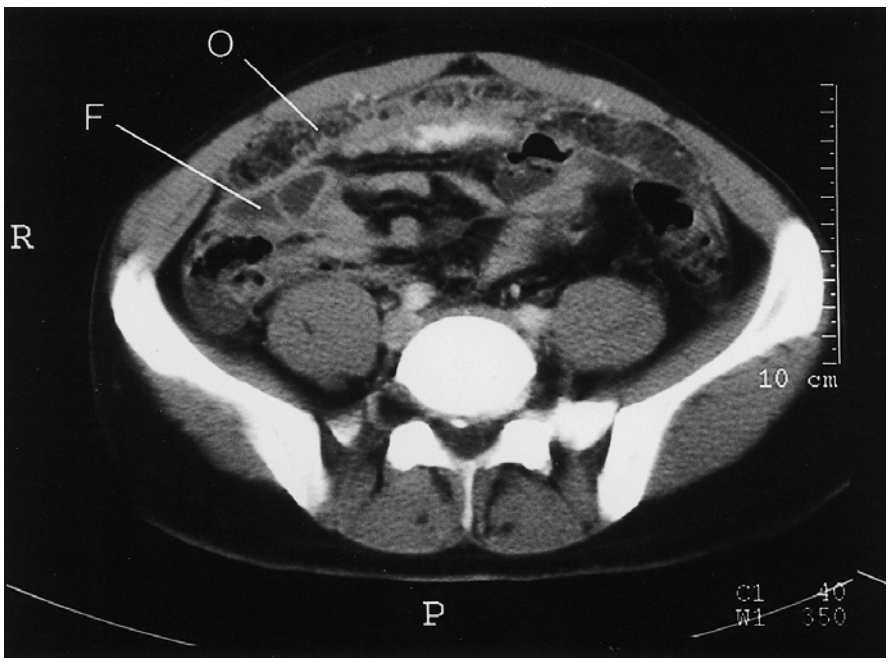

Fig 2. Abdominal computed tomography showing thickened omentum (O) and fluid adjacent to the terminal ileum (F) in abdominal tuberculosis.

Intra-abdominal abscesses

Intra-abdominal abscesses may occur at multiple anatomical sites within the abdomen. They are characterised by fever with localised tenderness, sometimes with a palpable mass. There is generally a leucocytosis with raised inflammatory markers. Microbiological investigations should include blood cultures (as there may often be associated bacteraemia), microscopy and culture of pus. The diagnosis is made by ultrasound or computed tomography (CT), at which time aspiration and/or therapeutic drainage of pus may be carried out, often avoiding the need for surgery. Bowel flora are frequently implicated, although there can be haematogenous seeding in a patient with infection elsewhere and associated bacteraemia (eg in endocarditis). Liver abscesses may be due to amoebiasis (see below). Empirical therapy should be started (Table 4) while awaiting culture results.

Amoebic liver abscesses

Liver abscesses (Fig 1) due to *E. histolytica* are associated with travel to warm climate countries, but most do not have a history of any preceding diarrhoeal illness. Amoebic serology is usually positive. Small amoebic abscesses may resolve on treatment with metronidazole alone but most will be aspirated for culture to exclude pyogenic abscess or prevent rupture in larger collections. Faecal microscopy may show *E. histolytica* cysts, but

this test is of low sensitivity, so even if this is negative, eradication of cysts is worthwhile in non-endemic areas with diloxanide furoate or paromomycin (metronidazole does not eradicate cysts from the bowel lumen).

Abdominal tuberculosis

Patients with abdominal tuberculosis (TB) usually have fever and weight loss, with abdominal pain of gradual onset over weeks or months. There may be a history of travel in areas where TB is common. Lymph nodes, ilio-caecal, liver and peritoneum are common sites (Fig 2). There may be evidence of TB elsewhere (most often pulmonary) and a positive tuberculin test, although this may be negative in the immunosuppressed, malnourished or those with severe disseminated disease. Diagnosis depends on obtaining samples of tissue or peritoneal fluid via laparoscopy or ultrasound- or CT-guided biopsy. These should be sent to the microbiology laboratory for microscopy and culture for mycobacteria, although histology demonstrating granulomata may also be helpful. Treatment consists of a standard six-month course of antituberculous chemotherapy, usually starting with quadruple therapy (rifampicin, isoniazid, pyrazinamide, ethambutol) for two months, followed by maintenance rifampicin and isoniazid for the remaining four months, unless the isolate shows resistance to these drugs.

Conflicts of interest

None.

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