

Severe infective gastroenteritis in a young man

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Gastroenteritis can manifest as different clinical syndromes from acute vomiting to bloody diarrhoea, which may suggest certain aetiologies, yet there can be considerable overlap.

As a GP working in both your own practice and the local emergency department, you commonly assess and treat patients with gastroenteritis from all age groups.

The case

One morning, a young man is wheeled straight from triage to an acute-care bed. The triage nurse quickly informs you that he is 32 years of age and presented with diarrhoea and abdominal pain, having recently returned from overseas. His blood pressure is 80/40 mmHg, heart rate 140 beats/minute, respiratory rate 24 breaths/minute and temperature 38.5°C.

You move to his bedside and initial assessment reveals he has dry mucous membranes and appears lethargic. He is severely dehydrated and has sepsis, so while you take a history, you insert an intravenous cannula and prescribe a one-litre bolus of normal saline. You send specimens for testing for venous blood gas levels, a full blood count, electrolyte and creatinine levels, liver function, lipase level and blood cultures.

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The history

You learn the man has travelled throughout Thailand for three weeks. His diarrhoea commenced on the day he returned home to Australia and has persisted for the past two days but he has not sought treatment until now.

He has had seven or eight episodes of watery diarrhoea per day that now contain blood and mucus. He has not vomited and is experiencing intermittent abdominal cramping. While overseas, he did not engage in any sexual activity. His travelling companion remains well. The illness has prevented the patient from returning to work as a chef.

He has no comorbidities and had an appendectomy at 21 years of age. He does not take any regular medications and has no known drug allergies. He is not immunocompromised and his family history is unremarkable.

Physical examination

On examination, you find that the patient's abdomen is soft but he has generalised tenderness. There is no percussion tenderness or rebound, and Murphy's sign is negative. He has no renal angle tenderness and no hepatomegaly or splenomegaly. Bowel sounds are present but hyperactive. The remainder of the examination is unremarkable.

The intravenous fluid bolus has finished and his blood pressure has improved slightly, having risen to 85/45 mmHg, but he remains tachycardic at 135 beats/minute. You ask for another litre of intravenous normal saline to be given as a bolus.

Test results and provisional diagnosis

The venous blood gas results have been returned and show the patient's pH is 7.35 and his lactate level is 2.6 mmol/L. His ECG

1. INDICATIONS FOR FAECAL MICROBIOLOGICAL TESTING¹

- Severe disease (see Box 2)
- Immunodeficiency
- Recent overseas travel
- Recent hospitalisation
- Antibiotic use in the preceding three months
- Public health reasons (aged care facility, institutional setting, food handler)

shows sinus tachycardia, and an erect chest x-ray is unremarkable. Your clinical assessment does not suggest an abnormality requiring surgical intervention. He does not have left lower-quadrant tenderness

that would suggest diverticulitis. The absence of vascular disease, atrial fibrillation or pain out of proportion to abdominal examination make you confident that he does not have ischaemic colitis. The rapid onset of symptoms and travel history make inflammatory bowel disease less likely; however, you acknowledge that this diagnosis can often be first recognised after travel. You think the most likely diagnosis is infectious gastroenteritis.

You are aware that faecal microbiological testing is not indicated for most patients you see with acute diarrhoea (Box 1).¹ Most cases are self-limiting, caused by viruses and will not return a positive culture. However, your patient has travelled overseas and is presenting with severe disease, so you order faecal microscopy, culture and

sensitivity testing (MCS), including for ova, cysts and parasites. You also order testing for *Clostridium difficile*.²

Most laboratories also screen with a faecal multiplex polymerase chain reaction (PCR), a nonculture-based method, which can test for multiple faecal pathogens, including bacteria, viruses and parasites (depending on the microbiological assay and laboratory service). This has the advantages of high sensitivity and specificity³ and rapid turnaround time (24 hours), but stool culture is still required to isolate bacteria not detected by the multiplex PCR and facilitate antibiotic susceptibility testing on bacterial pathogens.

The second one-litre bolus of fluid has been given, yet your patient remains tachycardic at 120 beats/minute and hypotensive at 90/50 mmHg. You give him another litre of intravenous fluid as a bolus. His blood pressure responds, rising to 100/60 mmHg, and his heart rate settles to 95 beats/minute. You commence more intravenous fluids at a rate of 250 mL/hour.

TABLE 1. SOME COMMON CAUSES OF INFECTIOUS GASTROENTERITIS⁵⁻⁷

	Viral	Bacterial	Protozoan
Consider whether these factors are present*	Self-limiting symptoms	Systemic symptoms	Prolonged (>14 days) [†]
	Noninflammatory diarrhoea [‡]	Inflammatory diarrhoea [§]	Immunocompromised patient
		History of travel	History of travel
Examples of possible infective organisms	Rotavirus	<i>Campylobacter jejuni</i>	Giardia
	Norovirus	<i>Salmonella</i> spp.	Cryptosporidium
	Enteric adenovirus	<i>Shigella</i> spp.	<i>Entamoeba histolytica</i>
	Astrovirus	<i>Clostridium difficile</i>	
		<i>Staphylococcus aureus</i>	
		<i>Bacillus cereus</i>	
		<i>Yersinia enterocolitica</i>	
		Noncholera <i>Vibrio</i> spp. (especially <i>Vibrio parahaemolyticus</i>)	
	<i>Escherichia coli</i>		
	<i>Vibrio cholerae</i>		

* May be considerable overlap. † Also consider noninfectious causes. ‡ Usually nonbloody, watery stool. § Usually bloody stool. || Usually noninflammatory.

Management and discussion

Many organisms may cause acute gastroenteritis, but most are viruses. These are typically transmitted via the faecal-oral route, although other routes of transmission are possible. Gastroenteritis can manifest as different clinical syndromes from acute vomiting to bloody diarrhoea, which may suggest certain aetiologies, yet there can be considerable overlap.⁴ Epidemiological features also suggest certain aetiologies (Tables 1 and 2).⁵⁻⁸

Your patient likely has travellers' diarrhoea, which affects 20 to 50% of travellers to developing countries and is more commonly caused by bacteria.^{1,6} Antibiotics are usually not required in nonsevere cases of bacterial gastroenteritis, and prescribing them can lead to harm (Box 2).^{1,6,7} The mainstay of treatment is fluid rehydration, which can be given orally or intravenously depending on clinical severity. Your patient has features of severe disease, so empirical antibiotic therapy and intravenous fluid therapy are warranted

(Box 2). Antibiotics should also be considered in immunocompromised patients. As this patient has recently returned from Southeast Asia, you decide to commence azithromycin because of high rates of quinolone resistance in that region.⁶

Loperamide is not recommended in your patient given the severity and bloody nature of the diarrhoea. However, in the absence of these features, it may be considered. The combination of loperamide and antibiotics has been shown to reduce the duration of symptoms in travellers' diarrhoea compared with antibiotics alone.^{6,7}

Progress

The patient is admitted. He remains normotensive and tolerates clear fluids. Relevant test results are shown in Table 3.

His liver function test results and lipase level are unremarkable. You attribute his acute kidney injury to hypovolaemia, and the hyponatraemia and hypokalaemia to gastrointestinal losses. These abnormalities return to normal levels with intravenous fluids and electrolyte replacement therapy.

The stool PCR test returns a positive result for *Campylobacter* spp. After three days his stool culture grows *Campylobacter jejuni* and sensitivities show resistance to ceftriaxone and ciprofloxacin but sensitivity to azithromycin. Azithromycin 500 mg orally is continued daily for a total duration of three days.

Outcome

On the third day of admission, the patient has improved significantly, is now eating

TABLE 2. EPIDEMIOLOGICAL AND HISTORICAL FEATURES OF COMMON ORGANISMS CAUSING INFECTIOUS GASTROENTERITIS^{5,7,8}

Epidemiological or historical feature	Possible aetiology to consider*
Travel to developing country	Enterotoxigenic <i>Escherichia coli</i> , <i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>Shigella</i> spp., <i>Entamoeba histolytica</i> and other protozoa, viruses
Contact in group settings (e.g. cruise ship, nursing home, hospital)	Norovirus
Outbreaks	Norovirus
Recent antibiotic use and/or hospitalisation	<i>Clostridium difficile</i>
Consumption of poultry or meats	<i>Campylobacter jejuni</i> , <i>Salmonella</i> spp., <i>E. coli</i> , <i>Yersinia enterocolitica</i> , <i>Bacillus cereus</i> , <i>Clostridium perfringens</i>
Consumption of rice	<i>B. cereus</i>
Consumption of shellfish or seafood	<i>Vibrio</i> spp.
Consumption of contaminated water or going camping	Giardia
Short incubation period (<6 hours)	Organisms with preformed toxins (<i>Staphylococcus aureus</i> , <i>B. cereus</i>)
Longer incubation period (>6–12 hours)	Organisms that make toxin after ingestion (e.g. <i>Vibrio</i> spp., <i>C. perfringens</i>) Organisms that directly invade bowel wall (e.g. <i>Shigella</i> spp., <i>C. jejuni</i>)

* This list is not exhaustive.

2. FEATURES OF SEVERE DISEASE IN GASTROENTERITIS AND EMPIRICAL THERAPY REGIMENS^{1,6,7}

Features of severe disease

- Severe dehydration
- Tachycardia
- Hypovolaemia
- High fever
- Severe abdominal pain and/or tenderness
- Bloody stools

Recommended empirical therapy for moderate to severe travellers' diarrhoea* (Australian Therapeutic Guidelines¹)

- Azithromycin 500 mg orally, daily for 2–3 days (especially if resistance suspected e.g. travel in Southeast Asia)[†] or
- Norfloxacin 400 mg orally, 12-hourly for 2–3 days[†] or
- Ciprofloxacin 500 mg orally, 12-hourly for 2–3 days

Recommended empirical therapy for suspected severe bacterial gastroenteritis (Australian Therapeutic Guidelines¹)

- Ciprofloxacin 500 mg orally, 12-hourly for 3 days or
- Norfloxacin 400 mg orally, 12-hourly for 3 days or
- Azithromycin 500 mg orally, daily for 3 days (if resistance suspected or quinolone contraindicated) or
- Ceftriaxone 2 g intravenously, daily for 3 days (if unable to tolerate oral form)

Potential unintended consequences of antibiotic use in nonsevere cases

- Increases resistance
- *Clostridium difficile* colitis
- Alters normal gut flora
- Prolongs carrier states
- Precipitates haemolytic uraemic syndrome[‡]

* Antibiotic therapy should be rationalised once susceptibilities are known.

[†] A single dose regimen with azithromycin (1 g) and norfloxacin (800 mg) can also be considered in the absence of fever and bloody stool.

[‡] In enterohaemorrhagic *Escherichia coli*, especially in children – antibiotics not recommended in children with bloody stools without fever.

TABLE 3. THE PATIENT'S RELEVANT CLINICAL CHEMISTRY AND FULL BLOOD COUNT RESULTS

Test	Test result
Haemoglobin	135 g/L
Platelets	430 x 10 ⁹ /L
White cell count	18.2 x 10 ⁹ /L
Neutrophils	13.1 x 10 ⁹ /L
Potassium	3.2 mmol/L
Sodium	130 mmol/L
Urea	13.8 mmol/L
Creatinine	148 µmol/L
Thick and thin blood film	No malarial parasites seen

and drinking well and his diarrhoea has almost completely resolved. You consider public health implications and consult state-based guidelines on reporting requirements for health practitioners and laboratories. You are still unsure so you contact your local public health authority.

You advise the patient to rest at home after discharge and, because he works in the food preparation industry, to not attend work until 48 hours after the complete resolution of diarrhoea. This period should also be 48 hours if the patient were to care for other people, including patients, the elderly or children. Twenty-four hours is recommended otherwise.

He returns to your practice in the community two weeks later and has had no further gastrointestinal symptoms or any other extra-intestinal complications (Box 3).^{4,9}

Conclusion

As with this patient, most adults will fully recover after an episode of acute infectious gastroenteritis. Initial management would have been similar irrespective of the causative agent; however, eliciting the patient's recent history and recognising symptoms of severe disease in this case

3. POTENTIAL COMPLICATIONS AFTER ACUTE GASTROENTERITIS^{4,9}

- Postinfectious irritable bowel syndrome
- Transient lactose intolerance (occurs more commonly in children)
- Reactive arthritis (rare with *Campylobacter jejuni* infection)
- Guillain-Barré syndrome (rare with *C. jejuni* infection)

were pivotal to the decision to administer antibiotic therapy and to the choice of antibiotic. MT

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References

1. Antibiotic Expert Groups. Therapeutic Guidelines: antibiotic. Version 15. Melbourne: Therapeutic Guidelines Ltd; 2014.
2. Gupta A, Khanna S. Community-acquired *Clostridium difficile* infection: an increasing public health threat. *Infect Drug Resist* 2014; 7: 63-72.
3. Reddington K, Tuite N, Minogue E, Barry T. A current overview of commercially available nucleic acid diagnostics approaches to detect and identify human gastroenteritis pathogens. *Biomol Detect Quantif* 2014; 1: 3-7.
4. LaRocque RC, Calderwood SB. Syndromes of enteric infection. In: Bennett JE, Dolin R, Blaser MJ, eds. *Mandell, Douglas and Bennett's principles and practice of infectious diseases*. 8th ed. Philadelphia: Elsevier Saunders; 2015.
5. Graves NS. Acute gastroenteritis. *Prim Care* 2013; 40: 727-741.
6. Leder K. Advising travellers about management of travellers' diarrhoea. *Aust Fam Physician* 2015; 44: 34-37.
7. Barr W, Smith A. Acute diarrhea in adults. *Am Fam Physician* 2014; 89: 180-189.
8. Hewison CJ, Heath CH, Ingram PR. Stool culture. *Aust Fam Physician* 2012; 41: 775-779.
9. Ghoshal UC, Ranjan P. Post-infectious irritable bowel syndrome: the past, the present and the future. *J Gastroenterol Hepatol* 2011; 26 Suppl 3: 94-101.

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