



During the first week of February this year, the South African health authorities reported two cases of cholera in people returning from Malawi. Since then, four more infections have been notified to the NICD. The third case was a household contact of the index patients, two infections have been acquired locally, and the origin of one case remains under investigation. This event highlights the ongoing risk that cholera may pose for travellers, and the community.

Epidemiology

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Cholera is an acute, secretory diarrhoeal disease caused by toxigenic strains of the Gram-negative bacterium Vibrio cholerae. Improved sanitation and access to safe water have largely eliminated indigenous cholera in high-income countries. However, cholera remains endemic in many middle and lower-income countries where both sporadic and 1 epidemic infections can occur. Upwards of four million cases of cholera are estimated to occur each year with approximately 150 000 deaths. The World Health Organization (WHO) estimates that officially-reported cases may 2 significantly under-represent the true number of infections . Cholera remains prevalent in many parts of Africa, Central America and Asia (Figure 1, below). The recent detection of two locally acquired cases is of great concern as South Africa has been regarded as being cholera-free.

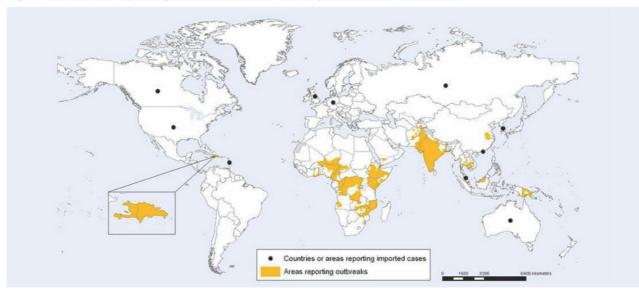


Figure 1. Countries reporting cholera outbreaks and imported cases of infection³



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Figure 1. In Africa, the number of reported cholera cases during January of this year was 30% higher than the whole of 2022.Malawi continues to experience a surge of infections and cases have been reported in neighboring Mozambique and Zambia, as well as in Burundi, Cameroon and the Democratic Republic of Congo. Ethiopia, Kenya and Somalia are also responding to outbreaks as a result of the extreme drought in the Horn of Africa, which has left millions in need of 4 humanitarian assistance.

Potential risk for travellers

Exposure to cholera is a potential risk for individuals and groups travelling to endemic countries. The degree of risk varies according to the area/s visited and duration of stay. In addition, cholera can be imported from areas in which it is endemic.

Vibrio cholerae is acquired by ingestion. A relatively large dose of organisms is necessary to establish infection. Factors that increase individual susceptibility include:

- use of proton pump inhibitors and antihistamines
- prior vagotomy
- type O blood group
- · concomitant Helicobacter pylori infection

Clinical disease and management

Clinical manifestations of cholera range from asymptomatic to varying degrees of diarrhoea, often with abdominal discomfort and nausea. Severe cholera (cholera gravis) is distinguished from other forms of gastroenteritis by profound and rapid loss of fluids and electrolytes. The stools are often described as having a "rice water" appearance, which can be accompanied by bile and mucus. The duodenum is the primary site of fluid secretion. Adult output can reach as much as 1 litre/hour, whereas in children it can be as much as 20 mL/kg/hour. The resulting hypovolaemia results in clinical signs of marked dehydration, poor tissue perfusion and lactic acidosis with hypokalaemia and hypocalcaemia. The primary principle of treatment is fluid resuscitation. Once an appropriate volume status has been achieved, antibiotic therapy can be initiated.

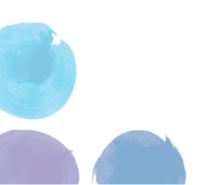
Antimicrobial therapy

A single daily dose of doxycycline 300 mg for two days has been shown to reduce disease duration. However, antibiotic resistance is common in certain parts of the world. Alternatives include azithromycin or ciprofloxacin. Prior to initiating antimicrobial therapy practitioners should consult their local microbiology specialists for updated information on the prevalence and distribution of drug-resistance and advice regarding antimicrobial therapy.

Infection control

Strict enteric precautions should be instituted when caring for individuals with cholera.





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Diagnosis

The clinical features of less severe cholera may be indistinguishable from enteritis caused by other travelassociated pathogens, including:

diarrhoeagenic E. coli infection; • salmonellosis; • shigellosis; • typhoid fever; • rotavirus and norovirus infections; • parasite infections including cryptosporidiosis, giardiasis and amoebiasis

Consideration should therefore be given to establishing a definitive aetiological diagnosis. Molecular testing of a stool sample by PCR is now the preferred procedure due to its rapidity and sensitivity. Where molecular tests are unavailable, bacteriological culture should be requested - in this setting the laboratory should be notified of the possible diagnosis of cholera as special procedures for bacterial enrichment and culture may be required.

The following additional tests may be required:

• **full blood count** - a leucocytosis without a left shift is frequently observed, together with a raised haematocrit due to haemoconcentration;

• **glucose** - hyperglycaemia may occur secondary to release of glucagon, adrenaline and cortisol due to hypovolaemia;

• metabolic tests - serum electrolytes, creatinine, urea and lactate.

References:

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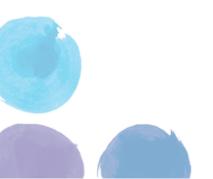
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