


A case of intercurrent shigellosis and rectal gonorrhoea in an acutely unwell febrile returned traveler

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Abstract: Both acute traveler's diarrhea and sexually transmitted infections are common causes of fever in the returned traveler, with the male sex corresponding to two-fold increased odds of a sexually transmitted infection (STI) diagnosis related to travel. *Shigella flexneri* is the most common cause of shigellosis in low- and middle-income countries, while within the men who have sex with men (MSM) population, outbreaks of *S. flexneri* 3a, *S. flexneri* 2a, and *S. sonnei* have been reported. We herein present a case of a febrile returned MSM traveler with a predominantly gastrointestinal presentation and proctocolitis whose microbiological work-up confirmed coinfection with *S. flexneri* and rectal gonorrhoea. Based on his travel history and epidemiologic risk factors, it is unclear if food- and waterborne shigellosis versus transmission via sexual contact was the major route of acquisition. This case highlights the broad differential for proctocolitis and the importance of consideration of intercurrent infections.

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Plain language summary

Acute bacterial diarrhea and a sexually transmitted infection causing fever in a returned traveler

We report a case of a healthy male traveler who acquired a foodborne bacterial traveler's diarrhea as well as sexually transmitted rectal gonorrhoea while traveling to Europe. He suffered from fever, diarrhea, and rectal pain before being fully treated with antibiotics.

Keywords: Enterobacteriaceae, gonorrhoea, sexually transmitted infection, shigellosis, traveler's diarrhea

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Introduction

Shigellosis is an acute diarrheal illness presenting as acute watery diarrhea to fulminant dysentery.^{1,2} It is spread via direct or indirect fecal-oral route, however, alternatively, via direct or indirect sexual contact.³ This includes anal or oral sex, anal play, and handling objects including sex toys, used condoms, barriers, or douching materials.³

Individuals at risk for shigellosis include children aged 1–4 in resource-limited settings, travelers to endemic areas, those experiencing homelessness, and children in daycare and their household contacts.² Men who have sex with men (MSM) with HIV infection are at increased risk due to factors, including compromised cell-mediated immunity and behavioral practices.⁴

This disease is caused by four species of *Shigella* bacteria: *S. sonnei*, *S. flexneri*, *S. dysenteriae*, and *S. boydii*. *S. flexneri* is the most common in low- and middle-income countries, whereas *S. sonnei* is the leading cause in high-income countries.² *S. dysenteriae* serotype 1 is associated with pandemics and is associated with severe disease. Incubation period is 1–4 days and typically self-resolves within 5–7 days.² Within *Shigella* isolates collected from the MSM population in the United Kingdom (UK), high rates of azithromycin resistance and outbreaks of *S. flexneri* 3a, *S. flexneri* 2a, and *S. sonnei* have been reported.^{5,6} Outbreaks of multidrug resistant (MDR) *S. sonnei* outbreaks have been documented in Montreal, and *S. flexneri* serotype 1 in Vancouver, within the Canadian MSM population.^{7,8}

Gonorrhea can cause infection of the pharynx, rectum, and urethra, and more rarely disseminated sites, and is caused by a Gram-negative diplococci: *Neisseria gonorrhoeae*.⁹ Gonorrhea rates have increased as much as 182% from 2010 to 2019 in Canada, with the most vulnerable age group in people less than 30 years of age.¹⁰ Gonococcal isolates collected from the MSM population are more likely to display antimicrobial resistance versus other groups.¹¹ Higher rates of gonorrhea infection in the MSM population are associated with risk factors, including multiple anonymous partners, oral sexual practices resulting in asymptomatic infection of the pharynx, and substance use or chemsex.¹¹

The Public Health Agency of Canada (PHAC) currently recommends treatment with a third-generation cephalosporin with either azithromycin or doxycycline.¹² Single-dose cephalosporin monotherapy regimens with higher dosing for gonococcal isolates with elevated minimum inhibitory concentrations (MICs) are recommended in other countries, including the UK and the USA.^{13,14}

The Committee to Advise on Tropical Medicine and Travel (CATMAT) identifies both acute traveler's diarrhea (10%–20%) and sexually transmitted infections (2%–3%) as common causes of fever in a returning traveler, with male sex corresponding to about a doubling in the odds of an STI diagnosis related to travel.^{15,16} CATMAT guidelines recommend consideration of empiric Ciprofloxacin in the treatment of adults with traveler's diarrhea for a duration of

3 days.¹⁵ The Infectious Disease Society of America (IDSA) recommends empiric treatment of infectious, bloody diarrhea with either fluoroquinolone or azithromycin.¹⁷

Sexually related gastrointestinal infections, including proctocolitis, have high rates of polymicrobial infection of up to 45.2%.¹⁸ These polymicrobial infections consist of enteric pathogens, parasites, and classic STIs. Up to 40% of cases of shigellosis in MSM individuals have been identified to have concomitant STIs, including rectal gonorrhea.¹⁹ Prior gonorrhea infection within the preceding year has been identified to be an independent risk factor for shigellosis within the MSM population living with HIV, with a 29.4-fold increased risk of acquisition.²⁰ Given these findings and overlapping symptoms, screening for parasitic, enteric, and STI infections is important to identify possible polymicrobial infections in this population.²¹

Shigella isolates can harbor plasmids for azithromycin resistance, such as the *mphA* and *ermB* genes, as well as beta-lactamases such as blaTEM-1, blaDHA-1, blaCTXM-15, and blaOXA-1.²² *Neisseria gonorrhoeae* ceftriaxone-resistance isolates are primarily due to harboring of the mosaic penA-60 allele.²³ Currently, there are no reports of documented horizontal gene transfer between *Shigella* species and *N. gonorrhoeae*. This could be of concern in the future with increasing rates of *N. gonorrhoeae* infection, existing ceftriaxone-resistant strains with mosaic penA-60 alleles, and the opportunity for horizontal gene transfer allowing for further means of ceftriaxone resistance in previously susceptible isolates during intercurrent infections.

This case report was conducted and reported in accordance with the CARE statement (Supplemental Material).²⁴ It highlights the importance of a broad differential diagnosis of proctocolitis. We herein present a case of a febrile returned traveler with a predominantly gastrointestinal presentation whose microbiological work-up confirmed coinfection with *S. flexneri* and rectal gonorrhea.

Case presentation

A 35-year-old man living with HIV, virally suppressed on bicitgravir-emtricitabine-tenofovir alafenamide and a CD4 count of 450 cells/mm³,

presented to the emergency department (ED) with an 11-day history of watery diarrhea with mucus, abdominal pain, and pain with defecation. He was febrile throughout this 11-day course, with a maximum temperature of 39.7°C on the day of presentation to the ED and notable tachycardia of 106 on examination.

His history was significant for travel to Portugal and Spain preceding his illness, with symptom onset on day 5 of travel. During his trip, he endorsed the consumption of raw seafood, local cuisine, unpasteurized dairy, and local water. His travel partners did not become sick during their trip. Prior to his trip, 7 days before symptom onset he endorsed an event with multiple new sexual partners where he engaged in condomless sex, and had taken doxycycline as pre-exposure prophylaxis prior to this event.

His presenting bloodwork demonstrated a normal leukocyte count of $5.4 \times 10^9/L$ (normal range 4.0–11.0 bil/L) and neutrophil count of $2.7 \times 10^9/L$ (normal range 2.0–7.5 bil/L). In the ED, a contrast CT of the abdomen and pelvis demonstrated acute proctocolitis. Blood and stool cultures were collected at this time. Rectal swabs were collected for molecular testing for gonorrhea, chlamydia, and Herpes simplex virus. He was empirically treated in the ED with 1 g of intramuscular Ceftriaxone and prescribed a 7-day course of Doxycycline 100 mg BID for gonorrhea and chlamydia. Given his febrile traveler's diarrhea, he was referred to our Tropical Disease Unit (TDU) via our Rapid Assessment of Diarrhea in Travelers clinic and evaluated in consultation the subsequent day.

Upon evaluation in the TDU, he reported persistent abdominal pain with loose stools despite empiric proctitis treatment. His fevers had resolved on the day of the presentation; however, endorsed ongoing rectal pain, tenesmus, and pain with defecation. He had begun taking the empiric course of doxycycline and was tolerating the medication well. On examination in the TDU, he was vitally stable with a blood pressure of 102/66 mmHg, pulse of 77 bpm, and temperature of 37.6°C orally. Physical examination was notable for abdominal tenderness in the left lower quadrant and palpable inguinal lymphadenopathy. There were no evident skin lesions or rashes, nor was there icterus.

Based on the totality of epidemiological risk factors, the differential diagnosis of this febrile gastroenteritis was broad, but most consistent with a typical traveler's diarrhea due to common enteropathogens such as *Campylobacter*, *E. coli*, salmonellosis, shigellosis, or yersiniosis. However, *Clostridioides difficile* infection, given the empiric antibiotics taken prior to travel, and more chronic or helminthic infections such as strongyloidiasis and schistosomiasis were considered given his prior travel history to endemic regions such as Southeast Asia, the Caribbean, and Mexico. A stool for ova and parasite examination, *C. difficile* toxin assay, and serologies for dengue virus, *Schistosoma*, and *Strongyloides* were ordered.

Following his initial ED visit, molecular testing returned positive for rectal *N. gonorrhoeae* infection. Despite empiric treatment of this infection in the ED, he continued to have ongoing but improving diarrhea, abdominal cramping, and fatigue at 18 days from symptoms onset and 7 days since empiric treatment for rectal gonorrhea, however, he did endorse the resolution of mucus discharge rectally. Stool culture from initial presentation in the ED returned positive for *S. flexneri*. His remaining infectious work-up—including for helminthiases—returned negative.

The patient had empirically received treatment for his rectal gonorrhea, warranting no further gonococcal-specific intervention. Given his persistent but improving diarrhea 18 days after symptom onset of the microbiological diagnosis of *S. flexneri* infection, he was provided a 3-day course of oral Ciprofloxacin 500 mg twice daily. Following initiation of this course, he had a complete resolution of his symptoms within 48 h of treatment.

Discussion

We present a case of intercurrent rectal gonorrhea infection and shigellosis in a returning traveler with a history significant for new sexual partners and as a member of the MSM population, virally suppressed HIV infection with a CD4 count of 450 cells/mm³, and multiple potentially significant dietary exposures while traveling including raw seafood, unpasteurized dairy, and street food. This case highlights the importance of maintaining a broad differential diagnosis of proctocolitis (Table 1), despite an early initial diagnosis, especially in the MSM population. The case further

Table 1. Comparison of select microbial causes of proctocolitis in febrile returned travelers.^{3,25–28}

Characteristic	<i>Shigella</i>	Gonorrhea	<i>Chlamydia trachomatis</i> lymphogranuloma venereum (LGV) serovars 1–3	Syphilis	Herpes simplex virus	<i>Campylobacter</i> spp.	Amebiasis
Incubation	1–4 days	5–10 days	3–30 days	10–90 days	2–12 days	2–4 days (up to 1–10 days)	2–4 weeks
Transmission	Fecal–oral route, consumption of contaminated food or water, anal sexual exposures	Anal sexual exposures (oral–anal, digital–anal, genital–anal)	Anal sexual exposures (oral–anal, digital–anal, genital–anal)	Anal sexual exposures (oral–anal, digital–anal, genital–anal)	Anal sexual exposures (oral–anal, genital–anal)	Consumption of contaminated foods, water, or dairy products More rarely, fecal–oral route	Fecal–oral route Anal sexual exposures
Pathophysiology	Bacteria reach large intestine, cross epithelium, and promote polymorphonuclear neutrophil leukocyte migration into epithelial lining resulting in clinical manifestations. Some strains produce <i>Shigella</i> enterotoxin 1 (ShET1) and SHET2 which induce fluid secretion into intestine. Shiga toxin, produced only by <i>S. dysenteriae</i> serotype 1, is cytotoxic and results in vascular lesions	Bacterium colonizes and infects anorectal mucosa resulting in local inflammation	Bacteria bind epithelial cells via heparin sulfate receptors, resulting in local inflammation. LGV serovars extend to regional lymph nodes resulting in a lymphoproliferative reaction	Chancere development in response to invasion of <i>T. pallidum</i> at site of inoculation.	Viral access via mucosa followed by replication in stratified squamous epithelium resulting in inflammation and tissue damage	Bacterial multiplication in human bile, invasion and destruction of epithelial cells. Flagella integral for chemotaxis and adherence, and some strains contain a heat-labile, cholera-like enterotoxin resulting in diarrhea	Ingestion of infective cysts, excystation in small intestine releasing motile trophozoites. Trophozoites migrate to large intestine and adhere and lyse colonic epithelium resulting in enteritis
Clinical features	Watery, bloody or mucoid diarrhea, fever, stomach pain, tenesmus	Anorectal pain, thick yellow mucopurulent discharge, tenesmus, bleeding, constipation	Rectal ulcers, anal discharge, bleeding, tenesmus, constipation, low-grade fevers, chills, malaise, myalgias, arthralgias, inguinal lymphadenopathy	Proctitis chancere, ulcer, or mass lesion, lymphadenopathy, pain with defecation, rectal bleeding, diarrhea, and tenesmus	Fever, tenesmus, constipation, radiculopathy, vesicles	Diarrhea, often bloody, abdominal pain, fever, and occasional nausea and vomiting More rarely, severe illnesses, including dehydration, bacteremia, symptoms mimicking acute appendicitis or ulcerative colitis	Gradual onset cramps, bloody or watery diarrhea, weight loss Extraintestinal manifestations – liver abscess

(Continued)

Table 1. (Continued)

Characteristic	<i>Shigella</i>	Gonorrhoea	<i>Chlamydia trachomatis</i> lymphogranuloma venereum (LGV) serovars 1–3	Syphilis	Herpes simplex virus	<i>Campylobacter</i> spp.	Amebiasis
Diagnosis of proctocolitis	NAAAT: PCR assay Culture: ID and AST from stool specimen or rectal swab	NAAAT: PCR of rectal swab Culture: rectal swab	NAAAT: PCR of rectal swab Culture: PCR for CT Identification and serovar confirmation (<i>ompA</i> and <i>pmpH</i> gene for serovar confirmation) Serology: antibody detection to chlamydial endotoxin	Serology: Various assays including CMIA, RPR, TP, PA)* Microscopy: Direct Fluorescence Microscopy of chancere exudate	NAAAT: PCR of lesions Culture: vesicular fluid Antibody testing: DFA Serology: glycoprotein G type-specific tests Microscopy: Tzanck smear for multinucleate giant cells from lesion scrapings	NAAAT: PCR, 16S rRNA sequencing, or whole-genome sequencing of stool sample Culture: stool specimen or rectal swabs, MALDI identification Antigen testing: stool specimens	Microscopy: stool ova and parasite examination for cysts and trophozoites NAAAT: PCR for species confirmation Antigen testing: ELISA for species confirmation Serology: extraintestinal amebiasis
Treatment	Often self-resolving in 5–7 days Abx for severe cases and in children: Azithromycin, Ciprofloxacin, Ceftriaxone, TMP-SMX	First line: Ceftriaxone 250–500 mg IM × 1 dose Second line: Gentamicin + Azithromycin, or Cefixime	First line, CT: Doxycycline 100 mg PO BID × 7 days First line, LGV: Doxycycline 100 mg PO BID × 21 days	First line: Benzathine Penicillin G 2.4 million units IM × 1 dose, versus weekly × 3 weeks	Symptom relief: oral analgesics, sitz baths First line, first episode: Acyclovir 200 mg PO 5 times per day × 5–10 days, Valacyclovir 1000 mg PO BID × 10 days, Famciclovir 250 mg PO TID × 5 days First line, recurrent episode: Acyclovir 200 mg PO 5 times per day × 5 days, Valacyclovir 500 mg PO BID or 1 g PO daily × 3 days, Famciclovir 125 mg PO BID × 5 days *May require IV acyclovir depending on severity	Often self-limiting, antibiotics indicated in severe, prolonged, or immunocompromised patient cases If indicated: Macrolides Fluoroquinolones	First line: Metronidazole or tinidazole, then iodoquinol or paromomycin Dosing, frequency, and regimen vary based on severity of disease
Prevention	Food and water precautions, good hand hygiene, if partner diagnosed with <i>Shigella</i> infection to wait at least 2 weeks after diarrhea ends to have sex	Partners within past 60 days to be referred for evaluation and treatment	Safe sex practices (barrier protection, cleaning sex toys and avoid sharing) Doxy PEP Empiric treatment of partners in past 60 days (Either Doxycycline x7 days or Azithromycin x1 dose)	Safe sex practices (barrier protection, cleaning sex toys and avoid sharing)	Safe sex practices (barrier protection, cleaning sex toys and avoid sharing)	Food and water precautions, good hand hygiene	Food and water precautions, good hand hygiene, avoid fecal exposure during sexual activity

*Serology may be non-reactive if early stages of infection.
ABX, antibiotics; CMIA, chemiluminescent microparticle immunoassay; NAAAT, nucleic acid amplification tests; O&P, ova, and parasite; PCR, polymerase chain reaction; RPR, rapid
plasma reagin; TP, PA, *Treponema pallidum* particle agglutination.

underscores the importance of even short courses of targeted antimicrobial therapy in prolonged shigellosis infection and offers an opportunity to review the current infection control guidelines surrounding shigellosis.

Shigellosis and rectal gonorrhea are both associated with abdominal pain, mucus discharge from the rectum, and fever.^{2,25} Both organisms can be transmitted sexually, with shigellosis also transmitted via direct and indirect fecal-oral routes. The incubation period for shigellosis is 1–4 days, with duration of illness that is usually only 5–7 days.² The incubation period for gonorrhea, on the other hand, is 5–10 days and can persist for months without treatment.²⁵ Our patient had a recent history of condomless intercourse with multiple new partners, increasing the risk of transmission for both shigellosis and gonorrhea and travel history concerning direct and indirect *Shigella* exposure. This case emphasizes the importance of obtaining an in-depth history, maintaining a broad differential, and close follow-up in patients with specific risk factors for multiple pathogens and more than one clinical syndrome.

In individuals living with HIV, the rates of Gram-negative bacterial enteric infections are at least 10 times higher than in the general population.²⁶ This rate declines with antiretroviral therapy and is inversely proportionate to the CD4 T lymphocyte count, with the greatest risk at counts < 200 cells/mm³. A decision regarding empiric therapy of gastroenteritis considers an individual's symptoms and their CD4 count, with counts of 200–500 cells/mm³ and severe diarrhea indications for a short course of antibiotics, compared to those with CD4 counts < 200 cells/mm³ in whom further extensive work-up should be performed in addition to longer courses of antimicrobial therapy.²⁶

Targeted antimicrobial therapy against *Shigella* spp. infection for individuals living with HIV, therapy is recommended.²⁶ In those with CD4 > 500 cells/mm³ where diarrhea resolves before culture confirmation, antimicrobial therapy may be withheld. In other cases where culture positivity is rendered during clinical illness, therapy should be offered ideally using ciprofloxacin for 7–10 days for isolates with MICs < 0.12 µg/mL.^{26,27} In cases where CD4 counts < 200 cells/mm³,

extended courses of antibiotics for up to 6 weeks may be warranted for recurrent or entrenched infections.²⁶ In our case, our patient had a CD4 count of 450 cells/mm³, and a 3-day course of ciprofloxacin was utilized given improving symptomatology over 2 weeks from onset and in accordance with CATMAT guidelines such intervention led to full resolution of symptoms within 48-h of initiation.¹⁵

Following treatment for shigellosis, the other management consideration in this patient was advice regarding infection prevention and control. *Shigella* spp. can be found in stool for up to 2 weeks after resolution of diarrhea in immunocompetent individuals.²⁶ CDC recommends waiting at least 2 weeks post-diarrhea resolution before engaging in any form of sexual intercourse given this risk of transmission.²⁶ Other groups recommend hand washing and avoiding sexual contact for 1 week after complete resolution of symptoms.²⁸ In our case, Toronto Public Health communicated infection control practices for our patients, however, it is important for healthcare providers to be aware of these recommendations and to discuss them with their patients to aid in outbreak prevention.

Conclusion

This case highlights a number of important points in regard to shigellosis in the MSM population and the need for consideration of intercurrent infections. Regardless of travel history, shigellosis should be considered in cases of acute gastroenteritis or proctocolitis given the range of potential transmission pathways. There is transmission risk, incubation, and symptomatic overlap between common STIs, especially rectal infections, and shigellosis; as such, the differential diagnosis should remain broad to account for intercurrent infections and reduce the risk of premature diagnostic closure. Antimicrobial therapy affords rapid clinical improvement compared to supportive management only, reduced carriage, and reduced potential for transmission. Clinical guidelines specific to a patient's underlying comorbidities and presentation exist, particularly for those living with HIV. Individuals diagnosed with shigellosis should be counseled at the time of diagnosis around behaviors that will reduce or prevent transmission.

Declarations

Ethics approval and consent to participate

Not applicable. Our institutions do not require ethical approval for reporting individual cases or case series when informed consent is provided by the patient.

Consent for publication

Verbal informed consent for publication was granted by the patient and recorded in the patient's chart during the medical encounter in accordance with PHIPPA.

Author contributions

Charlotte Fuller: Conceptualization; Data curation; Investigation; Writing – original draft; Writing – review & editing.

Ruchika Bagga: Data curation; Investigation; Writing – review & editing.

Ezra Bado: Data curation; Investigation; Writing – review & editing.

Syed Zain Ahmad: Data curation; Investigation; Writing – review & editing.

Andrea K. Boggild: Conceptualization; Data curation; Funding acquisition; Investigation; Project administration; Resources; Supervision; Writing – review & editing.

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Competing interests

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Availability of data and materials

All available data are presented in the text.

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Supplemental material

Supplemental material for this article is available online.

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