

# Severe Sepsis Due to *Chryseobacterium indologenes* in an Immunocompetent Adventure Traveler

 Genevieve McKew<sup>a,b</sup>

Department of Microbiology and Infectious Diseases, Concord Repatriation General Hospital, Sydney, Australia<sup>a</sup>; University of Sydney, Sydney, Australia<sup>b</sup>

***Chryseobacterium indologenes* is an environmental organism which is usually an opportunistic pathogen, most usually associated with nosocomial or device-related infections. This case, affecting a fit and well adventure traveler, demonstrates that it may be an agent of severe sepsis in otherwise healthy humans.**

## CASE REPORT

A 53-year-old man with no underlying medical conditions was admitted with septic shock 1 week after returning from hiking the Kokoda Track in Papua New Guinea. He presented with a 2-day history of dysuria and 1 day of prostration, fever, and rigors. He had initially become unwell 13 days prior to presentation, while hiking. He developed copious, frequent, watery diarrhea, vomiting, and malaise, for which he initiated treatment with norfloxacin at 400 mg twice daily for 7 days. The diarrhea resolved after 2 days, and he was able to continue hiking and complete the trek. He had no urinary symptoms at this time. He returned to his home city and returned to work. Four days after ceasing norfloxacin treatment, he developed urinary frequency. He became progressively more unwell and presented to his general practitioner, who referred him to a hospital the following evening. On presentation, he was in vasodilatory septic shock, febrile, and confused. His temperature was 41.5°C (106.7°F), his heart rate was 145 beats per minute, his respiratory rate was 24 breaths per minute, and his blood pressure was 89/55 mm Hg. He had ongoing urinary symptoms and was referred to the urology team for admission. He had no renal angle tenderness or prostatic tenderness on examination. His creatinine level was 1.69 mg/dl, he had a neutrophilic leukocytosis, and his venous lactate level was 5.1 mmol/liter. A computed tomography scan of the abdomen revealed presacral thickening and perirectal stranding consistent with proctitis. He was resuscitated with intravenous fluids in the emergency department, and persistent hypotension prompted transfer to the intensive care unit for vasopressor therapy. He was treated with ciprofloxacin and gentamicin for sepsis, presumably of urinary tract origin.

Cultures of urine and blood collected at the time of admission yielded a pure growth of bright yellow-pigmented colonies on 5% horse blood agar incubated in 5% CO<sub>2</sub> for 24 h. No other pathogens were detected on cultures or blood film, and stool microscopy revealed no inflammatory cells or parasites. Gram stain of the colonies showed Gram-negative rods. The organism was oxidase positive and spot indole positive and was tested and identified as *Chryseobacterium indologenes* by Vitek2 (bioMérieux) and API 20 NE (bioMérieux; biotype profile number 2610004, 99.5% probability, T 0.94). This was confirmed by partial 16S rRNA PCR, sequencing, and matching of the 427-bp fragment with BLASTN to sequences available in the GenBank database, with 100% query coverage and a 99% match, according to CLSI guidelines (1). Antibiotic susceptibility testing performed using automated broth microdilution and CLSI interpretive criteria for non-

fermentative Gram-negative rods (Vitek2; bioMérieux) gave an MIC for ciprofloxacin of 2 µg/ml (intermediate) and an MIC for trimethoprim-sulfamethoxazole of <20 µg/ml (susceptible). MICs for aminoglycosides, cephalosporins, and carbapenems were high (see Table 1). Blood cultures drawn more than 48 h after starting ciprofloxacin, when the patient was persistently febrile, also yielded this organism.

The patient remained febrile for more than 48 h, but confusion and hypotension resolved. He was admitted to the general ward. Ciprofloxacin treatment was continued. After 5 days, he was well enough for discharge. Due to the possibility of prostatitis, suggested by the history of urinary tract symptoms beginning several days after ceasing norfloxacin treatment and a mildly elevated prostate-specific antigen level (7.74 µg/liter), antibiotic treatment with trimethoprim-sulfamethoxazole was continued for 4 weeks. The patient remained well, and follow-up urine cultures were negative. A fasting glucose test result was normal.

*C. indologenes* is an oxidase-positive, non-glucose-fermenting Gram-negative bacillus which is found in the environment. Human infections usually occur in hospitalized patients, especially those who have received broad-spectrum antibiotics, and are often device associated or occur in patients who have had medical procedures or who have underlying medical conditions (2–7). This is the first reported case of severe sepsis from community-onset infection due to this organism in an otherwise healthy host. The patient became unwell after an initial diarrheal illness improved after quinolone treatment, with urinary symptoms and possible prostatitis. The biological sequence of events is unknown, but the organism was possibly acquired from a soil-contaminated hydration pack mouthpiece, causing a diarrheal illness which could have led to invasive infection with hematogenous seeding of the prostate. Another possibility is that the organism was inoculated through a breach in the skin. The patient did not notice any

Received 23 June 2014 Returned for modification 18 July 2014

Accepted 20 August 2014

Published ahead of print 27 August 2014

Editor: P. Bourbeau

Address correspondence to genevieve.mckew@sswhs.nsw.gov.au.

Copyright © 2014, American Society for Microbiology. All Rights Reserved.

doi:10.1128/JCM.01691-14

TABLE 1 Susceptibility testing (Vitek2) of *C. indologenes* isolate from blood

Antibiotic	MIC (mg/liter)	Interpretation <sup>a</sup>
Ampicillin	>32	R
Amoxicillin-clavulanate	>32	R
Cefazolin	>64	R
Cefepime	>64	R
Ticarcillin-clavulanate	>128	R
Meropenem	>16	R
Ciprofloxacin	2	I
Norfloxacin	4	S
Gentamicin	>16	R
Tobramycin	>16	R
Amikacin	>64	R
Cefoxitin	16	I
Nitrofurantoin	256	R
Trimethoprim	4	S
Trimethoprim-sulfamethoxazole	<20	S
Ceftazidime	32	R

<sup>a</sup> R, resistant; I, intermediate; S, susceptible.

significant cuts, but he was hiking through streams and wet terrain, and small breaches in the skin on the lower limbs may not have been noticed. The organism might have gained entry to the upper urinary tract hematogenously as a third possibility or, as a fourth possibility, by the more common route of urinary tract infection, via the urethra.

The Kokoda Track crosses the mountains in Papua New Guinea north of Port Moresby. It is popular with Australian tourists who wish to see the conditions experienced by Australian prisoners of war, who were marched over this mountain range during World War II. It is a difficult hike, with steep inclines and extremely rough terrain, requiring a high level of fitness (8, 9). The weather is extremely humid. During the patient's trek, temperatures were usually higher than 35°C (95°F), with frequent heavy rain and cold nights. He reported bathing in cold streams in his clothes daily and wore the same clothes for several days. He drank water from streams that had been sterilized in his hydration pack with an UV light source. Papua New Guinea is a resource-limited country (<http://databank.worldbank.org/data/home.aspx>), and little epidemiological data are available regarding bacterial infections. There are no previous reports of this infection occurring in a traveler returning from Papua New Guinea.

*C. indologenes* typically exhibits resistance to multiple antibiotics (2, 4, 6). The species is naturally resistant to aminoglycosides and possesses chromosomal metallo-beta-lactamases. Antibiotics with variable activity include the fluoroquinolones, tri-

methoprim-sulfamethoxazole, and rifampin. There are no established treatment recommendations. A case series of 16 patients with *C. indologenes* infections, all nosocomial and in patients with comorbidities, showed no clear relationship between antibiotic susceptibility and response to treatment: only 3 of the 16 patients received antibiotics to which the organism was susceptible, raising questions about the pathogenicity of this organism. One patient died of bacteremia-related mortality and did not receive an active antibiotic (2). This patient's isolate was susceptible only to trimethoprim-sulfamethoxazole, with intermediate susceptibility to ciprofloxacin. Serendipitously, the patient in this case received ciprofloxacin empirically for sepsis of urinary tract origin.

**Conclusion.** We report the first case of community-onset *C. indologenes* severe sepsis in an immunocompetent individual, indicating that the organism has the potential to be highly pathogenic. In patients who present with severe sepsis, a history of exposure to soil and water should be sought, and this organism should be considered a potential pathogen, noting its frequent resistance to many broad-spectrum antibiotics.

## REFERENCES

1. Clinical and Laboratory Standards Institute. 2008. Interpretive criteria for identification of bacteria and fungi by DNA target sequencing; approved guideline MM18-A. Clinical and Laboratory Standards Institute, Wayne PA.
2. Lin Y-T, Jeng Y-Y, Lin M-L, Yu K-W, Wang F-D, Liu C-L. 2010. Clinical and microbiological characteristics of *Chryseobacterium indologenes* bacteremia. *J. Microbiol. Immunol. Infect.* 43:498–505. [http://dx.doi.org/10.1016/S1684-1182\(10\)60077-1](http://dx.doi.org/10.1016/S1684-1182(10)60077-1).
3. Reynaud I, Chantepredrix V, Broux C, Pavese P, Croize J, Maurin M, Stah J-P, Jacquot C. 2007. A severe form of *Chryseobacterium indologenes* pneumonia in an immunocompetent patient. *Med. Mal. Infect.* 37:762–764. (In French.) <http://dx.doi.org/10.1016/j.medmal.2007.01.006>.
4. Cascio A, Stassi G, Costa GB, Crisafulli G, Rulli I, Ruggeri C, Iara C. 2005. *Chryseobacterium indologenes* bacteraemia in a diabetic child. *J. Med. Microbiol.* 54:677–680. <http://dx.doi.org/10.1099/jmm.0.46036-0>.
5. Green BT, Nolan PE. 2001. Cellulitis and bacteraemia due to *Chryseobacterium indologenes*. *J. Infect.* 42:219–220. <http://dx.doi.org/10.1053/jinf.2001.0822>.
6. Chou DW, Wu SL, Lee CT, Tai FT, Yu WL. 2011. Clinical characteristics, antimicrobial susceptibilities, and outcomes of patients with *Chryseobacterium indologenes* bacteremia in an intensive care unit. *Jpn. J. Infect. Dis.* 64:520–524.
7. Hsueh PR, Teng LJ, Yang PC, Ho SW, Hsieh WC, Luh KT. 1997. Increasing incidence of nosocomial *Chryseobacterium indologenes* infections in Taiwan. *Eur. J. Clin. Microbiol. Infect. Dis.* 16:568–574. <http://dx.doi.org/10.1007/BF02447918>.
8. Pattison DA, Walters TE, Seal E. 2011. Exercise-associated hyponatraemia on the Kokoda Track. *Med. J. Aust.* 194:247–248.
9. Seed CR, Coughlin JT, Pickworth AM, Harley RJ, Keller AJ. 2010. Relapsing vivax malaria despite chemoprophylaxis in two blood donors who had travelled to Papua New Guinea. *Med. J. Aust.* 192:471–473.