nosocomial transmission during that period (*9*). However, the emergence of MDR TB in regions of high HIV prevalence is relatively recent (*10*), and the cases described here suggest that increased vigilance for TB and MDR TB among migrating health care workers might be required.

Acknowledgments

We thank the families of patients who gave permission for this study and all those involved in data collection. We also thank Tim Brown, Vladyslav Nikolayevskyy, and Madeline Stone for the VNTR analysis and helpful comments and the staff of Public Health England National Mycobacterium Reference Laboratory for their assistance.

This study was supported in part by the Imperial College NHS Trust Biomedical Research Centre, National Institute for Health Research Health Protection Research Units (NIHR HPRU) in Healthcare Associated Infection and Antimicrobial Resistance and the NIHR HPRU in Respiratory Infections, both at Imperial College London in partnership with Public Health England. We received funding from the National Institute of Allergy and Infectious Diseases, National Institutes of Health, US Department of Health and Human Services, under contract no. HHSN272200900018C and grant no. U19AI110818. T.A. is a postdoctoral fellow of the Research Foundation–Flanders.

References

1. Brown TJ, Nikolayevskyy VN, Drobniewski FA. Typing *Mycobacterium tuberculosis* using variable number tandem repeat analysis. Methods Mol Biol. 2009;465:371–94. http://dx.doi.org/10.1007/978-1-59745-207-6_25
2. Gandhi NR, Moll A, Sturm AW, Pawinski R, Govender T, Lalloo U, et al. Extensively drug-resistant tuberculosis as a cause of death in patients co-infected with tuberculosis and HIV in a rural area of South Africa. Lancet. 2006;368:1575–80. http://dx.doi.org/10.1016/S0140-6736(06)69573-1
3. Cooke GS, Beaton RK, Lessells RJ, John L, Ashworth S, Kon OM, et al. International spread of MDR TB from Tugela Ferry, South Africa. Emerg Infect Dis. 2011;17:2035–7. http://dx.doi.org/10.3201/eid1711.110291
4. Ioerger TR, Koo S, No EG, Chen X, Larsen MH, Jacobs WR Jr, et al. Genome analysis of multi- and extensively-drug-resistant tuberculosis from KwaZulu-Natal, South Africa. PLoS ONE. 2009;4:e7778. http://dx.doi.org/10.1371/journal.pone.0007778
5. Li H, Durbin R. Fast and accurate short read alignment with Burrows-Wheeler transform. Bioinformatics. 2009;25:1754–60. http://dx.doi.org/10.1093/bioinformatics/btp324
6. Stamatakis A. RAxML-VI-HPC: maximum likelihood-based phylogenetic analyses with thousands of taxa and mixed models. Bioinformatics. 2006;22:2688–90. http://dx.doi.org/10.1093/bioinformatics/btl446
7. Walker TM, Ip CL, Harrell RH, Evans JT, Kapatai G, Dedicoat MJ, et al. Whole-genome sequencing to delineate *Mycobacterium tuberculosis* outbreaks: a retrospective observational study. Lancet Infect Dis. 2013;13:137–46. http://dx.doi.org/10.1016/S1473-3099(12)70277-3
8. Köser CU, Holden MT, Ellington MJ, Cartwright EJ, Brown NM, Ogilvy-Stuart AL, et al. Rapid whole-genome sequencing for investigation of a neonatal MRSA outbreak. N Engl J Med. 2012;366:2267–75. http://dx.doi.org/10.1056/NEJMoa1109910
9. Anderson LF, Tamne S, Brown T, Watson JP, Mullarkey C, Zenner D, et al. Transmission of multidrug-resistant tuberculosis in the UK: a cross-sectional molecular and epidemiological study of clustering and contact tracing. Lancet Infect Dis. 2014;14:406–15. http://dx.doi.org/10.1016/S1473-3099(14)70022-2
10. Abdool Karim SS, Churchyard GJ, Karim QA, Lawn SD. HIV infection and tuberculosis in South Africa: an urgent need to escalate the public health response. Lancet. 2009;374:921–33. http://dx.doi.org/10.1016/S0140-6736(09)60916-8

Address for correspondence: Graham S. Cooke, Imperial College, Jefferiss Laboratories, St. Mary's Campus, Praed St, London W21NY, UK; email: g.cooke@imperial.ac.uk

Fatal Bacteremia Caused by *Campylobacter gracilis*, United States

Takashi Shinha

Author affiliation: Vanderbilt University Medical Center, Nashville, Tennessee, USA

DOI: http://dx.doi.org/10.3201/eid2106.142043

To the Editor: *Campylobacter* species are well known to cause gastrointestinal infections in humans. However, extraintestinal illnesses caused by *Campylobacter* spp., including bacteremia, can also occur, primarily in immunocompromised persons (*1*). *Campylobacter gracilis* is a newly recognized species (*2*) that is commonly found in the oral flora and that has been associated with periodontal diseases and pleuropulmonary infections (*3–6*). Furthermore, a wide range of infectious etiologies caused by *C. gracilis* at different anatomic sites have been reported in the literature, suggesting its highly pathogenic potential (*7*,*8*). We describe a case of bacteremia due to *C. gracilis* complicated by pneumonia.

An 80-year-old man with a history of hypertension, hypertensive nephropathy, and chronic obstructive pulmonary disease (COPD) was in his usual health status when he began having worsening productive cough, fevers, and malaise; he sought health care 5 days later at Long Island College Hospital (Brooklyn, NY, USA). A heavy smoker who was noncompliant with his COPD treatment, he had frequent episodes of COPD exacerbation necessitating chronic maintenance with oral steroid therapy.

At physical examination, the patient appeared chronically ill and had mild respiratory distress. His temperature was 100.8°F, blood pressure 124/67 mm Hg, pulse 106 beats/min, respiration 22 breaths/min, and oxygen saturation 94% on room air. His heart sounds revealed tachycardia without murmurs, and his lung sounds disclosed scattered wheezing and rhonchi.

Laboratory studies revealed a leukocyte count of 14,400 cells/mm^3 (reference range 4,500–11,500) with 85% polymorphonuclear leukocytes, a hemoglobin level of 11.7 g/dL (reference range 14.0–18.0), and a platelet count of 174,000/mm^3 (reference range 150,000–450,000). His sodium level was 133 mmol/L (reference range 135–145), potassium 4.6 mmol/L (reference range 3.5–4.5), bicarbonate 30 mEq/L (reference range 22–28), urea nitrogen 118 mg/dL (reference range 9–23), and creatinine 5.2 mg/dL (reference range 0.7–1.3). A chest radiograph showed consolidation with large pleural effusion in the right lung.

He was empirically given vancomycin, cefepime, and azithromycin. Severe respiratory distress developed, and the patient died a few days later. Respiratory cultures at that time showed *Klebsiella pneumoniae* and *C. gracilis*. Blood cultures were positive for *C. gracilis*.

C. gracilis, originally known as *Bacteroides gracilis*, was transferred to the genus *Campylobacter* in 1995 after analysis of the cellular fatty acids, respiratory quinones, and proteins of *B. gracilis* and a comparison of them with the corresponding chemotaxonomic features of *Campylobacter* spp. (*2*). *C. gracilis* is a nonmotile, non–spore-forming, anaerobic gram-negative rod that uniquely requires formate and fumarate in its metabolism. *C. gracilis* primarily inhabits the gingival crevice and has been associated with a wide variety of periodontal diseases (*3*,*7*).

A study of 28 persons with chronic asymptomatic periradicular lesions showed *C. gracilis* in 6 (21.4%), including 2 (16.7%) of 12 who had acute apical periodontitis and 4 (23.5%) of 17 who had acute periradicular abscess (*4*). *C. gracilis* has also been isolated from other anatomic sites and has caused severe infections such as peritonitis, pneumonia, and bacteremia (*5*,*8*).

Our patient had *C. gracilis* bacteremia complicated by acute respiratory distress secondary to pneumonia. Although another gram-negative rod was isolated from the respiratory cultures, *C. gracilis* potentially played a major pathogenic role for this patient because of concomitant bacteremia that resulted in an unfavorable outcome. Pleuropulmonary infections with *C. gracilis* are not surprising because of the frequency of its detection in the human oral flora. In a study of 23 isolates of *C. gracilis* and their associated clinical diagnosis, 7 were from patients with lung abscess or empyema, and 2 were from those with aspiration pneumonia (*6*).

Campylobacter spp. are commonly associated with extraintestinal complications, including bacteremia, in immunocompromised hosts. In a study of 183 patients with *Campylobacter* bacteremia, the main underlying conditions were liver disease (39%) and cancer (38%). In that study, *C. fetus* was the most frequently identified species, found in 53% of the patients involved, followed by *C. jejuni*, *C. coli*, and *C. lari* (*1*). In another case report, *C. lari* bacteremia was described in a patient with multiple myeloma (*9*). Although uncommon, *C. gracilis* bacteremia has been reported in the literature (*8*).

Optimal antimicrobial drug treatment for *C. gracilis* remains to be established. Available antimicrobial susceptibility patterns in the literature have shown conflicting results (*5*,*10*). In 1 study, penicillin susceptibility was 67% and cephalosporin susceptibility was 67%–84% in 23 isolates of *C. gracilis* (*6*).

Further research is warranted to elucidate the mechanisms of pathogenicity and virulence of *C. gracilis*. Its pathogenic potential should not be underestimated because of the spectrum of disease, severity of infection, and its possible high frequency of antimicrobial drug resistance.

References

1. Pacanowski J, Lalande V, Lacombe K, Boudraa C, Lesprit P, Legrand P, et al. *Campylobacter* bacteremia: clinical features and factors associated with fatal outcome. Clin Infect Dis. 2008;47:790–6. http://dx.doi.org/10.1086/591530
2. Vandamme P, Daneshvar MI, Dewhirst FE, Paster BJ, Kersters K, Goossens H, et al. Chemotaxonomic analyses of *Bacteroides gracilis* and *Bacteroides ureolyticus* and reclassification of *B. gracilis* as *Campylobacter gracilis* comb. nov. Int J Syst Bacteriol. 1995;45:145–52. http://dx.doi.org/10.1099/00207713-45-1-145
3. Tanner A, Maiden MF, Macuch PJ, Murray LL, Kent RL Jr. Microbiota of health, gingivitis, and initial periodontitis. J Clin Periodontol. 1998;25:85–98. http://dx.doi.org/10.1111/j.1600-051X.1998.tb02414.x
4. Siqueira JF Jr, Rocas IN. *Campylobacter gracilis* and *Campylobacter rectus* in primary endodontic infections. Int Endod J. 2003;36:174–80. http://dx.doi.org/10.1046/j.1365-2591.2003.00636.x
5. Lee D, Goldstein EJ, Citron DM, Ross S. Empyema due to *Bacteroides gracilis*: case report and in vitro susceptibilities to eight antimicrobial agents. Clin Infect Dis. 1993;16 (Suppl 4):S263–5. http://dx.doi.org/10.1093/clinids/16.Supplement_4.S263
6. Johnson CC, Reinhardt JF, Edelstein MA, Mulligan ME, George WL, Finegold SM. *Bacteroides gracilis*, an important anaerobic bacterial pathogen. J Clin Microbiol. 1985;22:799–802.
7. Kamma JJ, Diamanti-Kipioti A, Nakou M, Mitsis FJ. Profile of subgingival microbiota in children with primary dentition. J Periodontal Res. 2000;35:33–41. http://dx.doi.org/10.1034/j.1600-0765.2000.035001033.x
8. Molitoris E, Wexler HM, Finegold SM. Sources and antimicrobial susceptibilities of *Campylobacter gracilis* and *Sutterella wadsworthensis*. Clin Infect Dis. 1997;25(Suppl 2):S264–5. http://dx.doi.org/10.1086/516234
9. Tauxe RV, Patton CM, Edmonds P, Barrett TJ, Brenner DJ, Blake PA. Illness associated with *Campylobacter laridis*, a newly recognized *Campylobacter* species. J Clin Microbiol. 1985;21:222–5.
10. Baron EJ, Ropers G, Summanen P, Courcol RJ. Bactericidal activity of selected antimicrobial agents against *Bilophila wadsworthia* and *Bacteroides gracilis*. Clin Infect Dis. 1993;16(Suppl 4): S339–43. http://dx.doi.org/10.1093/clinids/16.Supplement_4.S339

Address for correspondence: Takashi Shinha, Department of Pathology, Microbiology, and Immunology, Vanderbilt University Medical Center, TVC Ste 4514, 1301 Medical Center Dr, Nashville, TN 37232, USA; email: takashi.shinha@vanderbilt.edu