Five (62%) patients had received antimicrobial drugs before the infection. Drug therapy failed in 5 (62%) that had positive cultures during deoxycholate AMB (n = 4) or fluconazole (n = 1) therapy. Among the 7 patients with CVC-associated candidemia, 4 had the CVC removed; 3 of those survived. The 30-day all-cause mortality rate was 50%.

Our study showed a prevalence of 0.3% *C. haemulonii* among yeast isolates, which was much higher than previously reported (*4*). Older commercial methods are unable to correctly identify *C. haemulonii* species, contributing to this underestimation (*4*). More closely related species such as *C. auris*, mainly found in South Africa, Asia, and the Middle East, have been misidentified as *C. haemulonii* and *C. famata* by using older systems. Thus, matrix-assisted laser desorption/ionization–time of flight mass spectrometry and internal transcribed spacer rRNA sequencing are necessary to provide the correct identification (*5–7*).

The data we document suggest that patients with diabetes mellitus are more likely to have positive cultures for *C. duobushaemulonii* than for the 2 *C. haemulonii* species. Moreover, *C. duobushaemulonii* isolates have higher AMB MICs than the *C. haemulonii* species. As previously reported (*8*), echinocandins showed better in vitro activity than azole compounds.

In summary, we demonstrated that *C. haemulonii* species complex are critical pathogens of chronic lower extremity wounds and that fungemia by such species remains a rare event. The 30–day all-cause mortality rate among patients with candidemia was 50%, lower than previously reported in our institution (*9*) and other centers in Brazil (*10*). We believe that in cases of candidemia by *C. haemulonii* spp. that 1) empirical use of AMB or azole compounds should be avoided; 2) removal of CVC should be performed; and 3) antifungal susceptibility testing should be done to guide antifungal therapy.

References

1. Cendejas-Bueno E, Kolecka A, Alastruey-Izquierdo A, Theelen B, Groenewald M, Kostrzewa M, et al. Reclassification of the *Candida haemulonii* complex as *Candida haemulonii* (*C. haemulonii* group I), *C. duobushaemulonii* sp. nov. (*C. haemulonii* group II), and *C. haemulonii* var. *vulnera* var. nov.: three multiresistant human pathogenic yeasts. J Clin Microbiol. 2012;50:3641–51. http://dx.doi.org/10.1128/JCM.02248-12
2. Fujita SI, Senda Y, Nakaguchi S, Hashimoto T. Multiplex PCR using internal transcribed spacer 1 and 2 regions for rapid detection and identification of yeast strains. J Clin Microbiol. 2001;39:3617–22. http://dx.doi.org/10.1128/JCM.39.10.3617-3622.2001
3. Clinical Laboratory Standards Institute (CLSI). Reference method for broth dilution antifungal susceptibility testing of yeasts. Approved standard M27–A3, 3rd ed. Wayne, PA: National Committee for Clinical Laboratory Standards, The Institute; 2008.
4. Pfaller MA, Diekema DJ, Gibbs DL, Newell VA, Bijie H, Dzierzanowska D, et al. Results from the ARTEMIS Dros Inf Serv.K Global Antifungal Surveillance Study, 1997 to 2007: 10.5-year analysis of susceptibilities of noncandidal yeast species to fluconazole and voriconazole determined by CLSI standardized disk diffusion testing. J Clin Microbiol. 2009;47:117–23. http://dx.doi.org/10.1128/JCM.01747-08
5. Magobo RE, Corcoran C, Seetharam S, Govender NP. *Candida auris*-associated candidemia, South Africa. Emerg Infect Dis. 2014;20:1250–1. http://dx.doi.org/10.3201/eid2007.131765
6. Emara M, Ahmad S, Khan Z, Joseph L, Al-Obaid I, Purohit P, et al. *Candida auris* candidemia in Kuwait, 2014. Emerg Infect Dis. 2015;21:1091–2. http://dx.doi.org/10.3201/eid2106.150270
7. Kathuria S, Singh PK, Sharma C, Prakash A, Masih A, Kumar A, et al. Multidrug-resistant *Candida auris* misidentified as *Candida haemulonii*: characterization by matrix-assisted laser desorption ionization-time of flight mass spectrometry and DNA sequencing and its antifungal susceptibility profile variability by Vitek 2, CLSI broth microdilution, and Etest method. J Clin Microbiol. 2015;53:1823–30. http://dx.doi.org/10.1128/JCM.00367-15
8. Ramos LS, Figueiredo-Carvalho MHG, Barbedo LS, Ziccardi M, Chaves ALS, Zancopé-Oliveira RM, et al. *Candida haemulonii* complex: species identification and antifungal susceptibility profiles of clinical isolates from Brazil. J Antimicrob Chemother. 2015;70:111–5. http://dx.doi.org/10.1093/jac/dku321
9. Girão E, Levin AS, Basso M, Gobara S, Gomes LB, Medeiros EA, et al. Seven-year trend analysis of nosocomial candidemia and antifungal (fluconazole and caspofungin) use in intensive care units at a Brazilian University Hospital. Med Mycol. 2008;46:581–8. http://dx.doi.org/10.1080/13693780802004996
10. Colombo AL, Guimarães T, Silva LRBF, de Almeida Monfardini LP, Cunha AKB, Rady P, et al. Prospective observational study of candidemia in São Paulo, Brazil: incidence rate, epidemiology, and predictors of mortality. Infect Control Hosp Epidemiol. 2007;28:570–6. http://dx.doi.org/10.1086/513615

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Review of Cases and a Patient Report of Myiasis with Tracheostomy, Peru

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To the Editor: Myiasis is the infestation in humans of larvae of flies (order Diptera). These larvae can infect

skin, necrotic tissues, and natural cavities of living persons. Myiasis can be primary if it infects intact skin or secondary if it infects a previous injury. Depending on the degree of parasitism, myiasis may be obligatory (requiring a live host for parasite survival), facultative (developing in live or dead organic matter), or accidental (developing accidentally in an inappropriate host) (*1*). In South America, the species that most frequently cause myiasis are *Dermatobia hominis* and *Cochliomyia hominivorax*.

Factors contributing to development of myiasis are low socioeconomic status, unhealthy environments, advanced age, alcoholism, neurologic diseases, and lack of personal hygiene (*1,2*). Myiasis may occur different tissues, but reports of myiasis of the tracheal stoma are rare. We searched PubMed, MedLine, Lilacs, Scopus, and Google Scholar databases for scientific articles published in English or Spanish languages during 1990–2015 by using the search term "myiasis and tracheostomy." We found reports of 10 patients (Table).

We also report a case of tracheostomal myiasis in a 67-year-old man from Túcume, Peru. The patient had a history of esophageal tumor lesion with considerable airway stenosis related to upper esophageal cancer (stage III). Six months before onset of myiasis, he had respiratory difficulty caused by obstructed airway and underwent a tracheostomy and gastrostomy. When the patient was admitted to the emergency department of a hospital in Lambayeque, located ≈35 km from the patient's home, mobile larvae were present at the tracheostomy site, which also contained brown secretions with traces of blood and obvious signs of inflammation. A cervical abscess surrounded by necrotic tissue was visible, which, according to family members, developed after the larval infection. We manually removed the larvae and began treatment with ivermectin orally (1 dose, 200 µg/kg), ceftriaxone orally (2 g/d), and metronidazole intravenously (500 mg every 8 h). Three days after the patient started treatment, the tracheostomy tube was surgically removed for changing, and a large number of dead larvae were then observed and removed. The patient showed no signs of septicemia. He had slight relative eosinophilia (6%), but his hemoglobin and leukocyte levels were within reference ranges; the larvae would be unable to penetrate cells at these levels. The patient improved with no clinical symptoms of cervical abscess or evidence of phlogosis. He was discharged 9 days after admission, with a postdischarge treatment of oral metronidazole (500 mg every 12 h for 3 d).

Three specimens of larvae were sent to the hospital's parasitology laboratory, which identified the larvae as *C. hominivorax* stage L-3 (infection began with fly oviposition ≈6 days before admission; L-1, L-2, and L-3 are stages of larval development from hatching until pupation, requiring ≈7 days). The larvae were 10 mm × 3 mm and had a cylindrical, pale yellow body segmented with pigmented tracheal trunks visible in the last 4 posterior segments. Microscopic examination showed that the anterior end had a prominent jaw and segments with small bands of cuticular spines; the rear end had exposed spiracles, each with 3 straight grooves and open peritrematic membranes (reference *13* in the online Technical Appendix, http://wwwnc.cdc.gov/EID/article/22/3/15-1631-Techapp1.pdf).

The life cycle of *C. hominivorax* is similar to any other species in the Diptera order. Open wounds and body orifices (e.g., a tracheostomy) emitting odors from natural secretions are conducive for oviposition by flies and development of myiasis. A study from Brazil mentions that open wounds are the leading cause of development of the *C. hominivorax* parasite (*2*). Chronic extensive wounds are often infested by *C. hominivorax* (*2,5*).

Myiasis infection is concerning because it can lead to secondary infections such as *Escherichia coli*, *Serratia marcescens*, and *Enterococcus faecalis* (*6*). The infection is most dangerous when patients have concurrent conditions such as immunosuppression.

Table. Reports in the literature about myiasis associated with tracheostomy, by date of publication*

Country	Patient age, y/sex	Associated conditions	Fly species	Year of publication	Reference†
Canada	85/F	Comatose state for 2 mo	Unidentified	1993	(*3*)
Italy	57/M	Persistent vegetative state	*Lucilia caesar*	2006	(*4*)
Brazil	49/M	Neck carcinoma	*Cochliomyia hominivorax*	2011	(*5*)
India	78/M	Tracheostomy by car accident	*Chrysomya bezziana*	2011	(*6*)
Argentina	8/NA	Cerebral palsy	Unidentified	2012	(*7*)
India	52/M	Laryngeal cancer	*Musca domestica* (housefly)	2013	(*8*)
India	73/NA	Carcinoma supraglottis and diabetes	*Chrysomya bezziana*	2013	(*9*)
Turkey	86/F	Poor hygienic condition and tetraplegia	*Lucilia caesar*	2014	(*10*)
India	57/M	Proliferative ulcer on vocal cords and glottic stenosis	*Chrysomya bezziana*	2015	(*11*)
Italy	5/M	Werdnig-Hoffmann disease	*Sarcophaga argyrostoma*	2015	(*12*)
Peru	67/M	Esophageal cancer	*Cochliomyia hominivorax*		This study

*NA, not available.
†Sources: PubMed, Medline, Scopus, LILACS, and Google Scholar. References *11,12* are in the online Technical Appendix (http://wwwnc.cdc.gov/EID/article/22/3/15-1631-Techapp.pdf).

Treatment of myiasis involves manual removal of larvae and surgical debridement, in conjunction with ivermectin and systemic broad-spectrum antimicrobial drugs to prevent secondary infections (*1,2*). Treatment with ivermectin can kill the larvae (*1*; references *14,15* in the online Technical Appendix) and result in considerable reduction of larvae in infested wounds. Ivermectin has a broad antiparasitic spectrum that causes immobilization of parasites by inducing tonic paralysis of the parasite's muscles, mainly at the pharyngeal level, resulting in the death of the parasites by suffocation and starvation.

For the patient in this report, the single oral dose (0.2 mg/kg) of ivermectin was an effective treatment for myiasis. However, to control the underlying disease and prevent recurrences, ivermectin should be used with oral antimicrobial drugs and wound care when the wound has a high number of larvae, which are associated with bacterial infections (*4,5*).

For bedridden patients, patients with superficial wounds who live in myiasis-endemic areas, or patients who undergo a tracheostomy or have open wounds, health workers and caregivers should consider preventive care of wounds, which are risk factors for myiasis infection. This care consists of suitable wound dressing and proper personal and environmental hygiene.

Acknowledgments

References

1. Francesconi F, Lupi O. Myiasis. Clin Microbiol Rev. 2012;25: 79–105. http://dx.doi.org/10.1128/CMR.00010-11
2. Batista-da-Silva JA, Moya-Borja GE, Queiroz MMC. Factors of susceptibility of human myiasis caused by the New World screw-worm, *Cochliomyia hominivorax* in São Gonçalo, Rio de Janeiro, Brazil. J Insect Sci. 2011;11:14. http://dx.doi.org/ 10.1673/031.011.0114
3. Josephson RL, Krajden S. An unusual nosocomial infection: nasotracheal myiasis. J Otolaryngol. 1993;22:46–7.
4. Franza R, Leo L, Minerva T, Sanapo F. Myiasis of the tracheostomy wound: case report. Acta Otorhinolaryngol Ital. 2006;26:222–4.
5. Batista-da-Silva JA, Borja GEM, Queiroz MMC. Patient with tracheostomy parasitized in hospital by larvae of the screwworm, *Cochliomyia hominivorax.* J Insect Sci. 2011;11:163. http://dx.doi.org/10.1673/031.011.16301
6. Prasanna Kumar S, Ravikumar A, Somu L, Vijaya Prabhu P. Tracheostomal myiasis: a case report and review of the literature. Case Rep Otolaryngol. 2011;2011:303510. http://dx.doi.org/ 10.1155/2011/303510
7. Blejter J. Tracheostomy wound myiasis in a child: case report and review of the literature. Case Rep Pediatr. 2012;2012:317862.
8. Shakeel M, Khan I, Ahmad I, Iqbal Z, Hasan SA. Unusual pseudomyiasis with *Musca domestica* (housefly) larvae in a tracheostomy wound: a case report and literature review. Ear Nose Throat J. 2013;92:E38–41.
9. Hemanth V, Kumar CS, Manikandan D, Musarrat F, Preetham AP, Paulraj MG. An unusual cause of late tracheostomy bleed. Case Reports Clin Med. 2013;2:260–2. http://dx.doi.org/10.4236/ crcm.2013.24071
10. Kaya KH, Güneş S, Erdim İ, Koç AK, Avcı A, Kayhan FT. Tracheostomal myiasis in a female patient. Kulak Burun Boğaz Uygulamaları. 2014;2:132–4.

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Trends in Liver Transplantation in Hepatitis C Virus–Infected Persons, United States

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To the Editor: The Centers for Disease Control and Prevention and US Preventive Services Task Force recommend a one-time screening for hepatitis C virus (HCV) infection in adults born during 1945–1965 (birth cohort), a demographic group with a disproportionately high prevalence of HCV infection (*1,2*). However, some experts have warned against routine HCV screening of persons in the birth cohort, stating that this recommendation is based on unproven assumptions about the benefit of screening in reducing HCV-related mortality, given that only a minority of infected persons develop end-stage liver disease (ESLD) (*3*). To determine the relative effect of the birth cohort on HCV-related ESLD incidence in the United States, we analyzed trends in liver transplantation (LT) waitlist registrations and LT surgeries during 1995–2012. Using data from the United Network for Organ Sharing national registry, we evaluated birth cohort–specific (birth cohort vs. non–birth cohort) and etiology-specific (HCV vs. non-HCV) trends in LT waitlist registrations and LT surgeries performed in the United States during that 18-year period.

The proportion of HCV-infected persons born during 1945–1965 among all persons with LT waitlist registrations in the United States increased from 17.8% in 1995 to 35.2% in 2012 (Table). The highest proportion of LT