handling. No easy, definitive, and affordable test can demonstrate effectiveness before each use. Wearing may find the mask uncomfortable.

We encourage innovation to improve respiratory protection options. Future studies must be conducted to determine levels of protection achieved when naive users, following instructions, produce a similar mask from identical or similar raw materials. Research is needed to determine the minimal level of protection needed when resources are not available for N95 air-purifying respirators since the pandemic threat from H5N1 and other possible influenza strains will exist for the foreseeable future.

Virginia M. Dato,* David Hostler,* and Michael E. Hahn*

*University of Pittsburgh, Pittsburgh, Pennsylvania, USA

References

1. Occupational Safety and Health Administration. Guidance for protecting workers against avian flu. [cited 2005 Oct 23]. Available from http://www.osha.gov/dsg/guidance/avian-flu.html
2. National Institute for Occupational Safety and Health. 42 CFR Part 84 Respiratory protective devices. 1995 [cited 2005 Oct 23]. Available from http://www.cdc.gov/niosh/pt84abz2.html
3. Garner, JS. Guideline for isolation precautions in hospitals. The Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol. 1996;17:53–80.
4. Centers for Disease Control and Prevention. Laboratory performance evaluations of N95 filtering facepiece respirators, 1996. MMWR Morb Mortal Wkly Rep. 1998;47:1045–9.
5. World Health Organization Regional Office for the Western Pacific. Advice for people living in areas affected by bird flu or avian influenza. 2004 Nov 8 [cited 2005 Oct 22]. Available from http://www.wpro.who.int/NR/rdonlyres/04FA6993-8CD1-4B72-ACB9-E0EBDB3DC0CB1/0/Advice10022004rev08112004.pdf
6. Capps JA. Measures for the prevention and control of respiratory infections in military camps. JAMA. 1918;71:448–50.
7. Kool, JL. Risk of person-to-person transmission of pneumonic plague. Clin Infect Dis. 2005;40:1166–72.
8. Darling RG. Biological warfare and bioterrorism. Slides 47 and 48. [cited 2006 Mar 19]. Available from http://www.region-sem.org/IR/TG/JC/papers/123456/elr/Slides%20with%20Notes/Biological%20Warfare%20&%20Bioterrorism.pdf
9. Occupational Safety and Health Administration. Fit testing procedures (mandatory)—1910.134 App A. [cited 2006 Jan 21]. Available from http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=9780&p_text_version=FALSE#Appendix%20A
10. TSI incorporated. How to quantitatively fit test filtering-face piece respirators using a TSI Portacount Plus and N95-Companion (ITI-054) e2006. [cited 2006 Jan 21]. Available from http://www.tsv.com/AppNotes/appnotes.aspx?Pid=33&lid=445&file=iti_054

Address for correspondence: Virginia M. Dato, Center for Public Health Practice, Forbes Allies Center, 3109 Forbes Ave, Ste 210, Pittsburgh, PA 15260, USA; email: vdato@pitt.edu

Linguatuliasis in Germany

To the Editor: Pentastomids or tongue worms are a unique group of vermiform parasites, phylogenetically related to arthropods (1). Of the many pentastomid species, only a few, including Linguatula serrata, infect humans. The adult parasites are long, flat, or annulated and have 4 hooks surrounding a central mouth. Adult L. serrata inhabit the nasal passages and paranasal sinuses of wild and domestic canids, which serve as definitive hosts. Infective eggs containing larvae are discharged into the environment by nasopharyngeal secretions and are ingested by herbivores, the natural intermediate hosts. Humans can become dead-end intermediate hosts; visceral linguatuliasis then develops (2) if infective eggs are ingested. The liver is the organ most often involved (3–5), but the lung (4, 6, 7) or other organs (4, 8) may be affected. Parasites may also be found in lymph nodes. In the viscera, the primary 4-legged larva molts several times and eventually forms the legless nymph. Lesions due to Linguatula may be confused with malignancy, particularly in the lung (6).

We describe a recent infection with L. serrata in Germany in a patient who had pulmonary symptoms and in whom malignancy was suspected. The patient was a 39-year-old man of Russian origin who had been living in Germany since 1999. He was admitted to the hospital with weight loss, night sweats, chest pain, and coughing. He had been a smoker for 20 years, and his past medical history included pneumonia and sinusitis in 1989 during his military service at Lake Baikal, Russia. The patient had been living in a farmhouse in Karaganda, Kazakhstan, until he immigrated to Germany.

A chest radiograph and computed tomographic scan showed multiple, small lesions in both lungs. Malignancy was suspected, and a bronchoscopy was performed. Numerous granulomatus nodules were discovered. Thoracotomy was performed, and stringlike nodules on the pleural surface were resected. Except for a mild eosinophilia (7%, 500 cells/µL), the leukocyte count was normal. All other parameters, including C-reactive protein levels, angiotensin-converting enzyme, and tumor markers were normal. Histologic examination of the nodules showed a targetoid appearance with a sharp demarcation from the surrounding lung tissue by a thick fibrocollagenous capsule. In the center of the nodules, a transverse section (Figure, right inset) and a longitudinal section (Figure, main panel) of a parasite were visible. The parasite had a cutinuous cuticle 2.5 µm thick and cuticular
spines 20–30 µm long. The spines and the serrated aspect are characteristic for L. serrata, a pentastome. Ringlike structures in the body wall were interpreted as sclerotized openings, a key feature of pentastomes. In close contact to host tissue, a shed cuticle was visible and assigned to the previous instar larva. The biometric data of the parasite were comparable to those measured by others (6,9). Hooks, typical for the oral armature of pentastomes, were found by serial sectioning (Figure, left inset). Except for some subcuticular glands, the parasite’s inner organs were no longer distinguishable. The patient was initially treated with albendazole before the histologic diagnosis of linguatuliasis was established. Findings from magnetic resonance imaging of the abdomen were unremarkable, and no further lesions appeared during 12 months of followup. Intermittent cough and chest pain remained, possibly due to scar tissue and the remains of the nymphs.

At the beginning of the last century, visceral linguatuliasis of humans occurred frequently in Germany. In 1904 and 1905, among 400 autopsies in Berlin, 47 (11.8%) remains were infected with L. serrata (7). In contrast, reports of human infections are now rare. Our report is the first recent case description in Germany. Where the patient acquired the infection is unknown. L. serrata has a worldwide distribution. Recent cases have been reported from China (4) and Italy (6). An increasing number of infections can be suspected in the Western Hemisphere because of incremental travel to linguatuliasis-endemic areas. Humans are usually tolerant to nymphal pentastomid infections, and most patients are asymptomatic (4). The living nymph provokes little inflammation, whereas the death of the parasite leads to a prominent host response (2). Most findings of visceral linguatuliasis are made at autopsy (4,6), and the parasites are mainly located in the liver (3–5). Infection of the lung is rare (6,7). The nymphs in human granulomas are typically degenerated at the time of examination (3,6,9), but the cuticle with its associated structures remains visible for some time (2).

Histopathologic diagnosis is guided by the presence of remnants of the cuticle with sclerotized openings and by calcified hooks. Among pentastomids observed in humans, only L. serrata has prominent spines (2–4). In contrast to trematodes, the spines protrude from the cuticle and do not end in the body wall of the parasite. Diagnosis should be made etiopathologically, subetiopathologically, or presumptively on the basis of whether entire nymphs, cuticle-associated structures, or pearly lesions (“Linguatula nodules” [10]) with targetoid appearance are found (4). The differential diagnosis includes malignancies and tuberculosis because of the radiologic coinlike appearance. On histologic examination, one must distinguish between tissue-inhabiting diptera larvae, infections with metacestodes, trematodes, tissue filariids, and gnathostomiasis. Once diagnosis is established, no treatment is necessary (3) for the parasites will degenerate after some time, and no effective antiparasitic therapy exists. Avoiding contact with canine saliva and drinking water used by dogs or wild canids prevents this infection.

Dennis Tappe,* Ralf Winzer,† Dietrich W. Büttner,‡ Philipp Ströbel,*August Stich,‡ Hartwig Klinker,* and Matthias Frosch*
*University of Würzburg, Würzburg, Germany; †Bernhard-Nocht-Institute for Tropical Medicine, Hamburg, Germany; and ‡Medical Mission Hospital, Würzburg, Germany

References
1. Phylogenetic position of the Pentastomida and (pan) crustacean relationships. Proc Biol Sci. 2004;271:537–44.
2. Baird JK, Carey JC. Pentastomiasis. In: Connor DH, Chandler FW, editors. Pathology of infectious diseases. Stamford (CT): Appleton & Lange; 1997. p. 1671–4.
3. Hepatic granuloma in a man from North America caused by a nymph of Linguatula serrata. Pathology. 1988;20:198–9.
4. Pathological differentiation of suspected cases of pentastomiasis in China. Trop Med Int Health. 2002;7:166–77.

Figure. Linguatula serrata nymphs in lung tissue. Main panel shows the parasite’s serrated nature and the cuticular spines (magnification ×200, Masson trichrome stain). Right upper inset, pulmonary nodule with prominent fibrotic reaction and shed cuticle around 1 nymph (magnification ×200, Masson trichrome stain). Left lower inset, detailed view of 1 parasite hook (magnification ×630, hematoxylin and eosin stain).
We report this toxin-variant strain (binary toxin) as well as a deletion in genes encoding toxins A, B, and CDT (gene deletion was characterized by ribotyping (4) and by polymerase chain reaction (PCR) detection of genes that encode production of toxins A and B (5)). Toxin CDT was confirmed by amplifying the portion of the gene (cdtB) that encodes for the receptor-binding component of the toxin, according to a previously reported protocol (6). As a result, the isolate was classified as ribotype 027, toxinotype III (7), and was found to possess all 3 toxin genes. The tcdC gene deletion was also confirmed with PCR (8).

These results indicate that this canine isolate is indistinguishable from the major strain implicated in outbreaks of highly virulent CDAD around the world. According to the infection control practitioner at the hospital the dog visited, CDAD cases were occurring at increased frequency in the facility around the time the dog’s fecal specimen was collected. However, patient diagnosis was made solely through fecal toxin testing, and strains were not characterized. The facility has reported only sporadic cases of CDAD in the past few years. This is the first report of this human, epidemic strain of C. difficile in a dog. Many C. difficile strains isolated from animals, including dogs, are indistinguishable from strains associated with disease in humans (9).

To date, no study, including this one, has shown that interspecies transmission occurs; however, that possibility exists, as is becoming apparent with other pathogens, such as methicillin-resistant Staphylococcus aureus. The recurrent exposure of this dog to human healthcare settings suggests that the animal acquired this strain during visits to the hospital or long-term care facility, either from the healthcare environment or contaminated hands of human contacts. We recommend that future studies evaluating the dissemination of this strain and investigations of the movement of C. difficile into the community consider the role of animals.

Acknowledgments

We thank Joyce Rousseau for her assistance with culturing and identifying strains of C. difficile.

This work was supported by the Pet Trust Foundation of the Ontario Veterinary College.

Sandra L. Lefebvre,* Luis G. Arroyo,* and J. Scott Weese*

*University of Guelph, Guelph, Ontario, Canada

References

1. Centers for Disease Control and Prevention. Severe Clostridium difficile–associated disease in populations previously at low risk—four states, 2005. MMWR Morb Mortal Wkly Rep. 2005;54:1201–5.
2. Warny M, Pepin J, Fang A, Killgore G, Thompson A, Brazier J, et al. Toxin production by an emerging strain of Clostridium difficile associated with outbreaks of severe disease in North America and Europe. Lancet. 2005;366:1079–84.
3. Lefebvre SL, Waltner-Toews D, Peregrine AS, Reid-Smith R, Hodge L, Arroyo LG, et al. Prevalence of zoonotic agents in dogs visiting hospitalized people in Ontario: implications for infection control. J Hosp Infect. Epub 2006 Feb 5.
4. Barbut F, Lalande V, Burghoffer B, Thien HV, Grimprel E, Petit JC. Prevalence and genetic characterization of toxin A variant strains of Clostridium difficile among adults and children with diarrhea in France. J Clin Microbiol. 2002;40:2079–83.
5. Kato H, Kato N, Watanabe K, Iwai N, Nakamura H, Yamamoto T, et al. Identification of toxin A–negative, toxin B–positive Clostridium difficile by PCR. J Clin Microbiol. 1998;36:2178–82.
6. Stubbs S, Rupnik M, Gibert M, Brazier J, Duerden B, Popoff M. Production of actin-specific ADP-ribosyltransferase (binary toxin) by strains of Clostridium difficile. FEMS Microbiol Lett. 2000;186:307–12.