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Zoonotic Filariasis Caused by Novel *Brugia* sp. Nematode, United States, 2011

To the Editor: Zoonotic brugian filariasis is an incidental infection of humans with *Brugia* spp. nematodes

that primarily parasitize nonhuman vertebrates, rarely humans (1–3). In contrast to classical lymphatic filariasis caused by *B. malayi* and *B. timori*, which are found in Asia, most zoonotic *Brugia* infections have been reported from the northeastern United States (2,3) or South America (3). We report a case of symptomatic brugian infection in a New York City resident who had not traveled to the Eastern Hemisphere.

In 2011, a 53-year-old White man first noted tenderness and swelling behind his penis and in his right groin after having fallen 3 months earlier. The tenderness was relieved by non-steroidal antiinflammatory drugs, but the swelling continued; an oral antimicrobial drug, prescribed for presumed cellulitis, produced no improvement. At the time of examination, the patient had no fever or other signs or symptoms. Only a 3.0-cm × 3.0-cm firm, nonfixed right inguinal nodule without warmth or tenderness was noted. Laboratory findings were remarkable for total leukocytes of 6.4×10^9 , eosinophilia (12%, 600 cells/mm³), decreased hemoglobin level (10.0 g/dL), and low hematocrit of 31.2%. An excisional biopsy sample revealed intralymphatic adult nematodes with viable-appearing microfilaria (online Technical Appendix Figure, wwwnc.cdc.gov/EID/article/20/7/13-1654-Techapp1.pdf).

The patient had been born and raised in Champlain, Illinois, and had resided in the Bronx, New York, since 1979; he had no history of travel to filariasis-endemic regions. Characteristics of the adult worms and microfilaria were most consistent with those of *Brugia* spp., which was surprising because classical brugian lymphatic filariasis seems to be limited to Asia (*B. malayi*) and Indonesia (*B. timori*) (4,5). However, the adult filariae were smaller than expected for *B. malayi* or *B. timori* nematodes, prompting consideration of zoonotic filariasis (1,6). The adult worms and microfilaria seemed to be viable, although zoonotic *Brugia* spp. in histologic

sections often appear degenerated (1,2,6). The diameters of the adult worms were similar to those reported from South America (females 90–100 μm , males 50 μm) (7,8) rather than those from North America (females 35–75 μm , males 32–52 μm) (1). Peripheral blood was repeatedly negative for microfilaria. Serum sent to the Centers for Disease Control and Prevention (Atlanta, GA, USA) for ELISA testing for *B. malayi* anti-filarial IgG 4 showed optical density of 0.13, below the ELISA cutoff for filariasis.

Because micromorphologic information was not adequate for species identification, paraffin-embedded biopsy specimens were submitted for molecular testing. Genomic DNA extracted from paraffin-embedded tissue with the QIAamp DNA–formalin-fixed, paraffin-embedded tissue procedure was amplified by using the primer sets DiBu-F(5' GCTAGATATGCTACCAACAAA-3')/ITS1 R(5'-CTCAATGCGTCTGCAATTCGC-3') and BuF2-(5-CATTATGCTAGATATGCTACCAAC-3')/ITS1-R.

The products were fractionated on 2% agarose gel and stained with ethidium bromide. The internal transcribed spacer (ITS) 1 PCR product (182 bp) was automatically sequenced by using the same primers used for PCR. Lasergene software (DNASTAR, Madison, WI, USA) was used to align the sequences obtained with *Brugia* spp. sequences deposited in GenBank; detailed sequence comparison identified the isolate as a novel *Brugia* (Nematoda: Onchocercidae) species closely related to *B. pahangi* and *B. malayi* (Figure). The ITS-1 sequence was submitted to the EMBL Nucleotide Sequence Database (accession no. HE856316).

Removal of an affected lymph node without additional treatment is often considered sufficient treatment for zoonotic filariases. However, for the patient reported here, persistence of inguinal swelling prompted a repeat biopsy 4 months later; the specimen again demonstrated reactive follicular hyperplasia, although no parasites were seen. Because the patient's initial

clinical signs and subsequent persistent adenopathy were reminiscent of unilateral lymphadenitis, lymphangitis, and induration that are typical of *B. malayi* or *B. timori* filariasis, and the microfilariae in the original biopsy sample appeared to be viable, we empirically prescribed a standard dosage of oral doxycycline for 6 weeks, followed by single doses of ivermectin at 400 $\mu\text{g}/\text{kg}$ and 800 mg albendazole. The patient has been well, without further adenopathy or eosinophilia, for >2 years. Because adult filariae can live for >10 years, the place of acquisition cannot be stated with certainty.

The prevalence of zoonotic infection with *Brugia* spp. nematodes is unknown. Many reported cases are asymptomatic or diagnosed incidentally during evaluation for persistent adenopathy (1–3). Conversely, differentiation of zoonotic from classical filariasis is unlikely in disease-endemic areas; most cases published since the initial 1962 case report (1) occurred in the United States. Most case-patients were from the Northeast,

JQ327146	--AAAAAAAAAAAAAAAAAGACATACAAAAAATTATATATATATATATTATAGTAATAATAA	58
EU373624	---AAAAAAAAAAAAAAAAAGACATACAAAAAAT--TATATATATATATATTATAGTAATAATAA	56
EU419348	--AAAAAAAAAAAAAAAAAGACATACAAAAAAT----ATATATATATATTATAGTAATAATAA	54
EU373632	-----AAAAAAAAAAAAACATACAAAAAAGT---TATATATATATATTATAGTAATAACAA	50
EU373630	-----AAAAAAAAAAAAACATACAAAAAAGT---TATATATATATATTATAGTAATAACAA	50
AY621469	AAAAAAAAAAAAAAAAAACATACAAAAAAGT---TATACATATATATTATAGTAATAACAA	57
EU419351	-----AAAAAAAAAAAAACATACAAAAAAGT---TATACATATATATTATAGTAATAACAA	53
HE856316	---AAAAAAAAAAAAAACATACACATAAATTTG--TATATATATATAATAGTAATAACAA	57
EU373647	-----AAAAAAAAAAAAACATACAAAAAAGT---TATATATATATATTATAGTAATAACAA	51
	*****..*****.*:** *** *****:***** **	
JQ327146	T-AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAGGAACG	117
EU373624	T-AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	115
EU419348	TAAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	114
EU373632	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	107
EU373630	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	107
AY621469	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	115
EU419351	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	111
HE856316	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGGCTCATGGATCGATGAAGAACG	114
EU373647	T---AAAATTTTTTTAACTCTTAGCGTGGATCACTTGTCTCATGGATCGATGAAGAACG	108
	* ***:*****	
JQ327146	CAGCTAGCTGCGA	130 (92.13%)
EU373624	CAGCTAGCTGCGA	128 (92.80%)
EU419348	CAGCTAGCTGCGA	127 (94.35%)
EU373632	CAGCTAGCTGCGA	120 (95.83%)
EU373630	CAGCTAGCTGCGA	120 (95.00%)
AY621469	CAGCTAGCTGCGA	128 (93.60%)
EU419351	CAGCTAGCTGCGA	124 (94.31%)
HE856316	CAGCTAGCTGCGA	127
EU373647	CAGCTAGCTGCGA	121 (94.21%)

Figure. Pile-up of partial ribosomal DNA sequences from *Brugia* NY strain (HE856316) and from other related *Brugia* spp. strains and clones, *B. malayi* BM28 (JQ327146), *B. malayi* C27Cat5 (EU373624), *B. pahangi* C61CAT5 (EU419348), *B. pahangi* C14Cat6 (EU373632), *B. pahangi* C7Cat6 (EU373630), *B. pahangi* Bp-1 (AY621469), *B. pahangi* C46CAT5 (EU419351), and *B. pahangi* C27Cat7 (EU373647). Boxes indicate the *Brugia* NY strain (HE856316); asterisks (*) indicate conserved residues; periods (.) indicate nucleotide changes; colons (:) indicate nucleotide changes just in the *Brugia* NY isolate; hyphens (-) are included in the sequences to maximize the comparisons among the 9 DNA molecules. Italicized numbers in parentheses indicate the percentage of similarity with the *Brugia* NY isolate.

including New York (8 cases), Massachusetts, Pennsylvania, Connecticut, and Rhode Island (3 cases each) (1,2); single cases have been identified in Michigan, Ohio, North Carolina, Oklahoma, New Jersey, Louisiana, Florida, and California (1,2). Four other cases have been reported: 3 in South America (Colombia, Brazil, Peru) (3,7,8) and 1 in Africa (Ethiopia) (9). Only a few *Brugia* species have been identified, including *B. leporis*, found in rabbits in the northeastern United States (1,10); *B. beaveri*, found in raccoons and bobcats in the southern United States; and *B. guyanensis*, found in coati-mundi and other vertebrates in South America (8). Definitive identification with molecular techniques will better identify causative species and help clarify many of the ecologic and epidemiologic questions surrounding zoonotic filarial infections.

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***Candida auris*– Associated Candidemia, South Africa**

To the Editor: We noted the report by Chowdhary et al. (1) and report *Candida auris* as a causative agent of candidemia in South Africa, with an estimated prevalence of 0.3% (N.P. Govender et al., unpub. data). First isolated in 2009, *C. auris* is an emerging species associated with clinical disease (2–6). We analyzed 4 isolates submitted to the National Institute for Communicable Diseases (Johannesburg, South Africa) from 4 patients with candidemia who had been admitted to different public- and private-sector hospitals from October 2012 through October 2013.

Identification of the isolates was undertaken by using ChromAgar *Candida* medium (Mast Diagnostics, Merseyside, UK), Vitek-2 YST (bioMérieux, Marcy l'Etoile, France), API 20C AUX (bioMérieux), and sequencing of internal transcribed spacer (ITS) and D1/D2 domains of the ribosomal RNA gene (7), followed by microbroth dilution susceptibility testing (8). All isolates were misidentified as *C. haemulonii* and *Rhodotorula glutinis* by Vitek-2 YST and API 20C AUX assays, respectively (Table).

Similar to the findings of Chowdhary et al., all isolates assimilated *N*-acetyl-glucosamine (1). With the use of the CBS-KNAW database, pairwise sequence alignment of ITS region showed 99% sequence homology to Kuwait isolates, and alignment of D1/D2 domain showed 98% homology to the Kuwait/India isolates (9). In a neighbor-joining phylogenetic tree based on ITS sequences, South Africa isolates formed a cluster with India and Kuwait isolates (online Technical Appendix Figure, <http://wwwnc.cdc.gov/EID/article/20/7/13-1765-Techapp1.pdf>).