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Septic Arthritis Caused by *Streptococcus suis* Serotype 5 in Pig Farmer

To the Editor: *Streptococcus suis* primarily infects pigs, but >700 human infections have been reported (*1*). Cases in human occur mainly in persons who have contact with pigs; these infections are most frequently reported in Southeast Asia (*1*). In humans, *S. suis* most often causes meningitis, but endocarditis, pneumonia, toxic shock–like syndrome, and septic arthritis have also been reported (*1–3*).

S. suis is classified into serotypes on the basis of the polysaccharide capsule. Among pigs, many serotypes cause severe infections, but nearly all human cases have been attributed to serotype 2 (*1,3*). Other serotypes have been isolated from humans only in a few cases: meningitis caused by serotype 4 (*2*); fatal bacteremia caused by serotype 16 (*4*); sepsis caused by serotype 24 (*5*); bacteremia, meningitis, and endocarditis caused by serotype 14 (*6–8*); and spontaneous bacterial peritonitis caused by serotype 5 (*5*). Here, we report a case of septic arthritis caused by *S. suis* serotype 5.

The patient was a 65-year-old pig farmer who had cut his hand at work; he had not noted cases of severe illness among his pigs. He had a history of benign hyperplasia of the prostate gland, and 1 year before the current illness, he received a diagnosis of right-sided coxarthrosis, for which radiographic imaging showed grade II changes, loss of cartilage, and subchondral sclerosis. One week after the patient cut his hand, his right hip became increasingly painful, and he sought treatment at a hospital. On examination, the trochanter major region was tender (not noted at previous examinations), and passive movement of the hip was painful. Blood test results showed a slight elevation of C-reactive protein (CRP), to 31 mg/L (reference <5 mg/L). The symptoms were interpreted as trochanteritis, and treatment with nonsteroidal anti-inflammatory medication was instituted. The next day, the patient returned to the hospital with worsened pain and was admitted. He had a temperature of 37.7°C and a heart rate of 80 beats/min; blood test results showed a leukocyte count of 11.2×10^9 cells/L and CRP of 127 mg/L. Radiologic images of the hip were unremarkable, but ultrasonography-guided joint puncture showed pus and blood in the synovial fluid. Cultures were secured, and gram-positive cocci in short chains were noted in all blood culture bottles and in the synovial fluid culture. Treatment with intravenous cefotaxim was started.

Microbiological diagnosis of *S. suis* infection was made on the basis of colony morphology, a weak reaction with Lancefield anti-D antiserum, and a score of 2.31 according to matrix-assisted laser desorption/ionization–time of flight mass spectrometry (Biotyper version 3.0 software; Bruker Daltonics, Bremen, Germany). On the fourth day after admission, treatment was changed to benzylpenicillin (3 g 3×/d). The pain from the hip gradually declined, and CRP peaked at 337 mg/L on the fifth day after admission. On the seventh day after admission, treatment was changed to oral penicillin (2 g 3×/d) and was continued for 6 weeks.

At follow-up 6 months after the initial illness onset, the impairment in the patient's hip movement had worsened. Radiologic imaging showed necrosis of the femoral head, and the patient underwent total hip replacement surgery. During surgery, no signs of synovitis were noted, and 5 intraoperative cultures were negative. The procedure was completed without complications, and the patient's symptoms resolved.

The *S. suis* isolate from the patient was determined to be serotype 5 by Statens Serum Institut (SSI;

Hillerød, Denmark) by agglutination with latex beads and type-specific serum and by microscopic determination of capsule swelling with type-specific serum (SSI Diagnostika, Hillerød, Denmark), according to the manufacturer's instructions. These methods gave concordant results. Etests (bioMérieux, Solna, Sweden) demonstrated that the isolate was sensitive to all antimicrobial drugs tested; MIC was 0.125 mg/L for cefotaxim and 0.016 mg/L for benzylpenicillin. The isolate was tested for known virulence-associated genes *sly*, *mrp*, and *epf* with PCR, as described (*9*). PCR fragments of predicted sizes were obtained with primer hybridizing to *sly* and *mrp* but not with primers hybridizing to *epf*. A serotype 2 isolate (kindly provided by Susanne Sauer at SSI) was used as a positive control for the *epf* primers.

S. *suis* is an emerging human pathogen, but reports of human infections caused by serotypes other than serotype 2 remain scarce. This case demonstrates that S. *suis* of serotype 5, which is a serotype routinely isolated from deceased pigs (*10*), can cause invasive infections in humans. The course of the described infection was relatively favorable, and the patient did not show signs of a systemic inflammatory response syndrome or of meningitis. Preexisting osteoarthritis of the right hip might have had diminished local defenses, thereby enabling colonization of the hip area by bacteria that had entered the bloodstream through the wound on the patient's hand. The isolate we recovered possessed *sly* and *mrp* genes, which encode the virulence-associated suilysin and muraminidase-released proteins, but clearly other factors are also of importance for determining the virulence of individual S. *suis* isolates.

The patient gave his informed consent to the writing of this article.

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Bartonella henselae and *B. koehlerae* DNA in Birds

To the Editor: Bartonellosis, a globally emerging vector-borne zoonotic bacterial disease, is caused by hemotropic, gram-negative, aerobic, facultative intracellular *Bartonella* spp. (*1*). Of the 30 *Bartonella* species/subspecies, 17 have been associated with human infections (*2,3*). Each species has a reservoir host(s), within which the bacteria can cause intraerythrocytic bacteremia with few or no clinical signs of illness (*1,3*); the bacteria are transmitted by hematophagous arthropod vectors (*1*). Various *Bartonella* spp. have been identified in domestic and wild animals, including canids, deer, cattle, rodents, and marine mammals (*1,4*). *Bartonella* DNA from the blood of loggerhead sea turtles (*Caretta caretta*) has been PCR amplified and sequenced (*5*); the fact that *Bartonella* DNA was found suggests the possibility that persistent blood-borne infection can occur in nonmammals and that the host range for *Bartonella* spp. may be larger than anticipated.

Growing evidence suggests that wild birds play key roles in the maintenance and movement of zoonotic pathogens such as tick-borne encephalitis virus and *Borrelia* and *Rickettsia* spp. (*6–9*). *Bartonella grahamii* DNA was amplified from a bird tick in Korea (*10*). The substantial mobility, broad distribution, and migrations of birds make them ideal reservoir hosts for dispersal of infectious agents.

To investigate whether birds might be a reservoir for *Bartonella* spp., we screened 86 birds for the presence of *Bartonella* spp. DNA.

The primary study site was a residential backyard in Morehead City, North Carolina, USA (34°43.722′N, 76°43.915′W). Of the 86 birds screened, 78 (16 species) were captured by mist net during March 2010–June 2012 and 8 (3 species) were injured birds that were to be euthanized (Table). Each bird was examined for external abnormalities and ectoparasites, weighed, measured, and tagged with a US Geological Survey–numbered band. A blood sample (0.10–0.25 mL) was collected from each bird by using a 1-mL insulin syringe with a 28-gauge × 1.27-cm needle. Blood remaining after preparation of blood smears was added to an EDTA tube and frozen (–80°C) until processed. Blood smears were examined for hemoparasites. Research was conducted under required state and federal bird banding permits and with the approval of the North Carolina State University Institutional Animal Care and Use Committee.

Before DNA was extracted from the samples, 10 µL of blood was diluted in 190 µL of phosphate-buffered saline. DNA was automatically extracted by using a BioRobot Symphony Workstation and MagAttract DNA Blood M96 Kit (QIAGEN, Valencia, CA, USA). *Bartonella* DNA was amplified by using conventional *Bartonella* genus PCR primers targeting the 16S–23S intergenic spacer region: oligonucleotides, 425s (5′-CCG GGG AAG GTT TTC CGG TTT ATCC-3′) and 1,000as (5′-CTG AGC TAC GGC CCC TAA ATC AGG-3′). Amplification was performed in a 25-mL reaction, as described (*3*). All PCR reactions were analyzed by 2% agarose gel electrophoresis. Amplicons were sequenced to identify the *Bartonella* sp. and intergenic spacer region genotype. To compare sequences with those in GenBank, we identified bacterial species and genotypes by using Blast version 2.0 (http://blast.ncbi.nlm.nih.gov/Blast. cgi). DNA extraction and PCR-negative controls remained negative throughout the study.

Results are summarized in the Table. None of the screened birds were anemic, but 5 were PCR positive for *Bartonella* spp. (3 for *B. henselae* and 2 for *B. koehlerae*). *B. henselae* was amplified from 2 Northern Mockingbirds (*Mimus polyglottos*) and 1 Red-winged Blackbird (*Agelaius phoeniceus*) (GenBank accession no. KC814161). The DNA sequences were identical to each other and had 99.6% (456/457 bp) sequence similarity with *B. henselae* San Antonio 2 intergenic spacer region genotype (GenBank accession no. AF369529). *B. koehlerae* was amplified from a Red-bellied Woodpecker (*Melanerpes carolinus*) and a Common Loon (*Gavia immer*) (GenBank accession no. KC814162). The DNA sequences were identical to each other (404/404 bp) and to GenBank sequence AF312490. Lice (Mallophaga order) were found on 5 Boat-tailed Grackles (*Quiscalus major*), but no ectoparasites were observed on *Bartonella* spp.–positive birds. Hemoparasites (*Haemoproteus* and *Plasmodium* spp.) were detected in 7 of 86 birds, indicating exposure to hematophagous ectoparasites, but hemoparasites were not detected in the *Bartonella* spp.–positive birds. No bacteria were visualized in *Bartonella* PCR–positive blood smears.

Bartonella spp. are increasingly associated with animal and human illnesses; thus, the identification of reservoirs and increased understanding of *Bartonella* spp. disease ecology are of public health importance. Our finding of 2 pathogenic species not previously reported in birds has expanded the potential sources for zoonotic infection.

There is growing evidence that migratory birds serve as reservoirs

Table. *Bartonella* species detected in birds*			
Bird common name	Bird species	No. birds positive/no. total	*Bartonella* sp.
House sparrow	*Passer domesticus*	0/28	
Boat-tailed grackle	*Quiscalus major*	0/15	
Mourning dove	*Zenaida macroura*	0/12	
Herring gull†	*Larus argentatus*	0/6	
House finch	*Carpodacus mexicanus*	0/5	
Blue jay	*Cyanocitta cristata*	0/3	
Song sparrow	*Melospiza melodia*	0/2	
Northern cardinal	*Cardinalis cardinalis*	0/2	
Northern mockingbird	*Mimus polyglottos*	2/2	*B. henselae* SA2
European starling	*Sturnus vulgaris*	0/2	
Red-winged blackbird	*Agelaius phoeniceus*	1/1	*B. henselae* SA2
Brown thrasher	*Toxostoma rufum*	0/1	
Tufted titmouse	*Baeolophus bicolor*	0/1	
Red-bellied woodpecker	*Melanerpes carolinus*	1/1	*B. koehlerae*
Common grackle	*Quiscalus quiscula*	0/1	
Common loon†	*Gavia immer*	1/1	*B. koehlerae*
Red-headed woodpecker	*Melanerpes erythrocephalus*	0/1	
Brown pelican†	*Pelicanus occidentalis*	0/1	
Collared dove	*Streptopelia decaocto*	0/1	

*SA2, San Antonio 2 intergenic spacer region genotype.
†Euthanized.