the manuscript. Written consent for publication was obtained from the patient’s wife.

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Streptococcus suis
Meningitis without History of Animal Contact, Italy

To the Editor: Streptococcus suis, a major swine pathogen worldwide, is emerging as a zoonotic agent capable of causing a variety of serious infections in swine as well as in persons exposed to pigs or to pork products. These infections include meningitis, septicemia, pneumonia, endocarditis, arthritis, and septic shock (1,2). Despite recent outbreaks among persons in China, S. suis disease in humans is a rare, probably underdiagnosed infection that usually occurs as sporadic cases (1,2). Persons in close occupational or accidental contact with pigs or pork products and those who eat uncooked or undercooked pork may be at higher risk than others. However, most infected persons are likely healthy carriers, and S. suis is believed to induce overt disease (especially meningitis) in only some circumstances (2). We describe a case of S. suis meningitis in a 68-year-old man from Sardinia, Italy, who had no reported contact with swine, other animals, or any animal products; the patient also had cancer, which was discovered incidentally during the workup.

In November 2007, the patient was hospitalized with a 48-hour history of fever, headache, nausea, and general malaise. Physical examination showed impaired consciousness, nuchal rigidity, and a temperature of 39.5°C. Laboratory findings were 20,700 leukocytes/mm³ with 92% neutrophils, glucose 95 mg/dL, and C-reactive protein 375 mg/L. Cerebrospinal fluid (CSF) analysis demonstrated 240 leukocytes/μL with 80% polymorphonuclear cells, glucose 24 mg/dL, and protein 277 mg/dL. A computed tomography scan of the head showed no abnormal findings. Gram stain of CSF showed gram-positive cocci, mostly in pairs (Figure).

Empirical therapy consisted of intravenous ceftriaxone (2 g twice a day) and oral chloramphenicol (2 g once a day). On day 5, α-hemolytic streptococci were isolated from CSF on sheep blood agar and identified as S. suis by using APIStrep (bioMérieux, Marcy l’Etoile, France). Serotyping, performed by slide agglutination with specific antisera (Statens Serum Institute, Copenhagen, Denmark), identified the isolate as serotype 2.

Antimicrobial drug–susceptibility testing, performed according to guidelines of the Clinical and Laboratory Standards Institute (www.clsi.org), indicated susceptibility to penicillin, ceftriaxone, chloramphenicol, levofloxacin, and vancomycin and resistance to erythromycin (MIC >128 mg/L) and tetracycline (MIC 16 mg/L). Erythromycin resistance was constitutive and was mediated by theerm(B) determinant; tetracycline resistance was mediated by tet(W). Multilocus sequence typing (http://ssuis.mlst.net) assigned the S. suis isolate to sequence type (ST) 1.

The patient, a retired welder, denied any recent occupational or even occasional contact with swine or other animals and had no history of eating raw or undercooked pork. The patient’s condition improved; chloramphenicol was discontinued on day 10, but the 14-day course of ceftriaxone was completed. On day 6, the patient
became afebrile but had dizziness and deafness; a formal audiology evaluation on day 9 showed severe bilateral sensorineural high-frequency hearing loss (−80 dB) that improved after a short course of dexamethasone. However, the patient was not discharged because of the lung mass found on initial chest radiograph. Computed tomography scan, bronchoscopy, and histopathologic findings led to diagnosis of the mass as an advanced-stage squamous cell carcinoma.

The meningitis had common and uncommon features. The common features were hearing loss, a typical outcome of S. suis meningitis independent of early antimicrobial drug administration (1,2); serotype 2, the most frequent and virulent serotype in swine and in humans (1,2); ST1, belonging to the ST1 complex, strongly associated with S. suis meningitis isolates (2,3); and erm(B)-mediated erythromycin resistance, widespread in this species (4). The uncommon features were tetracycline resistance mediated by tet(W), increasingly detected in gram-positive and in gram-negative bacteria (5) but never previously reported in S. suis or in other major streptococcal pathogens, where common determinants are tet(M) and tet(O); and lack of evidence for recent contact with swine, other animals, or swine (pork) products.

Two previous cases of human S. suis meningitis in Italy (6,7) and other recent cases from Europe (8,9) were related to occupational exposure. However, the patient reported here also had cancer, and malignancy has been indicated as a predisposing factor for the development of severe S. suis disease in humans (2). These findings appear to be consistent with the recent suggestion of new epidemiologic patterns of infection caused by this organism (2). S. suis may become an opportunistic pathogen in persons who are under stress or who have immune-deficiency, and it has been increasingly isolated from mammalian species other than pigs, from birds, and from the environment. As also discussed in a recent survey (10), the possibility cannot be excluded that a patient with S. suis infection may be unaware or have no memory of previous exposure to animals. Alternatively, because asymptomatic carriage of S. suis has been documented in humans (2) and is believed to contribute to its transmission (10), the possibility should also be considered that the infection may be a reactivation, possibly favored by malignancy, of latently colonizing S. suis.

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Figure. Gram-positive cocci, mostly in pairs, in cerebrospinal fluid from a 68-year-old man with Streptococcus suis meningitis. Magnification ×1,000.
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Equine Herpesvirus 
Type 9 in Giraffe 
with Encephalitis

To the Editor: Herpesviruses have been isolated from many mam- 
 mals. Herpesvirus infection in natural hosts is often mild and is usually 
 followed by a latent infection; however, cross-species herpesvirus infections 
 cause severe and fatal diseases. Equine herpesvirus (EHV)–1 causes abortion, 
 respiratory disease, and, occasionally, neurologic disorders in horses. EHV-1 
 infection is usually limited to equine species, although it has also been found 
 in other species (1), in which it causes fatal encephalitis. Recent sequence 
 analyses suggested that the equine herpesviruses isolated in the United 
 States from onagers (Equus hemio-
 nius), Grevy’s zebras (E. grevyi), and 
 Thomson’s gazelles (Gazella thomso-
 ni) are a subtype or variant of EHV-1 
 (2). With respect to epizoology, the 
 nonequine animals affected by EHV-1 
 or EHV-1–related virus were kept in 
 enclosures adjacent to those of zebra 
 species (Grevy’s or Burchell’s).

Another EHV-related virus was isolated from 2 Thomson’s gazelles 
 that had encephalitis and were kept with zebras (3). The virus was lat-
 ter found to be a new type of EHV, EHV-9, although it was serologically 
 cross-reactive with EHV-1 (3). Re-
 cently, neutralizing antibodies against 
 EHV-9 were found among Burchell’s 
 zebras in the Serengeti ecosystem (4).

A herpesvirus was recently iso-
 lated from a reticulated giraffe (Gi-
raffa camelopardalis reticulate) with 
 neurologic symptoms; the giraffe 
 was from a zoo in the United States 
 (5). Nonsuppurative encephalitis was 
 found by histopathologic examination 
 of the giraffe brain. Several Burchell’s 
 zebras that were apparently healthy 
 and later determined to be seroposi- 
 tive for EHV-1 were housed in the same 
 pen as the giraffe. The isolated virus 
 was identified by PCR and a monoclo- 
 nal antibody assay as EHV-1 (5). In 
 the present study, we analyzed 4 gene 
 sequences of the giraffe herpesvirus 
 to show its relatedness to EHV-1 and 
 EHV-9.

We amplified portions of 4 genes 
 from giraffe herpesvirus DNA by PCR. 
 The DNA polymerase catalytic sub-
 unit (open reading frame [ORF] 30) 
 gene was amplified by using herpes-
 virus universal primers (6). The genes 
 for glycoprotein B (gB) (ORF33), 
 glycoprotein 2 (gp2) (ORF71), and 
 glycoprotein D (gD) (ORF72) were 
 amplified by using primers specific for 
 EHV-9. The ORF73 primers were gp2-F 
 (5′-GGCACTATGCTCCATAGTAC 
 TGGTTGCTG-3′) and gp2-R (5′- 
 AAATATCTCAGGCGGAGAAG 
 TGGAAAGTG-3′). The ORF71 prim-
 ers were gp2-F (5′-CCCGTTGAGT 
 AGTCTTGGTAGAAGTCTA-3′) and 
 gp2-R (5′-GCCACAAGCCTGAC 
 TAAAGCCAAGGAGTTCC-3′). 
 The ORF72 primers were gD-F 
 (5′-TTTCAACACCCTGTGGC 
 GTGTCGAGAA-3′) and gD-R (5′- 
 ATCTCCTCCACGGCAAGACTT 
 TAAGCCCGT-3′). The amplified 
 products were used as templates for 
 direct sequencing (Dragon Genom- 
 ics, Mie, Japan). The sequences were 
 edited with Phred, Phrap, and Consed 
 (www.phrap.org/phredphrapconsed. 
 html), and the phylogenetic trees were 
 constructed with PHYLIP (2,7). Ac-
 cession numbers of the sequences 
 (submitted to the DNA Data Bank of 
 Japan) are given in the Figure.

We used PCR to amplify a part of the 
 gB gene of the giraffe herpesvirus, 
 and we used EHV-1 specific primers 
 for sequencing. However, we could 
 not obtain amplicons (data not shown). 
 Therefore, the more conserved gene, 
 ORF30, was sequenced. The sequence 
 of the 1,066-bp segment of the giraffe 
 herpesvirus ORF30 gene was 99.5% 
 identical to EHV-9 and 94.6% identi-
 cal to EHV-1, which indicates that the 
 giraffe herpesvirus was most closely 
 related to EHV-9. Therefore, EHV-9 
 ORF33–specific primers were used to 
 amplify the corresponding region of 
 the giraffe herpesvirus. The sequence 
 of the giraffe herpesvirus ORF33 was 
 98.8% identical to EHV-9 and 95.9% 
 identical to EHV-1. Also, the sequence 
 of the other envelope glycoproteins 
 (ORF71 and ORF72) of the giraffe her-
 pesviruses were 99.8% and 99.6% iden-
 tical to EHV-9 and 91.6% and 96.3% 
 identical to EHV-1. A phylogenic tree 
 of maximum likelihood showed that 
 EHV-9 and the giraffe virus formed a 
 genetic group that was apparently dis-
 tinguished from other genetic groups 
 of EHV (Figure).

Herpesviruses have caused clin-
 ical disease in zoo animals, including a 
 case of EHV-9 infection in Thomson’s 
 gazelles (3) and a recently described 
 endotheliotropic betaherpesvirus in-