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 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 antiserum (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 the erm(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...
mediated by features were tetracycline resistance in this species (erythromycin resistance, widespread previously reported in negative bacteria (tetracycline-resistant gram-positive and in gram-negative bacteria (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 mammals. 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 hemionus), Grevy’s zebras (E. grevyi), and Thomson’s gazelles (Gazella thomsoni) are a subtype or variant of EHV-1 (2). With respect to epizootiology, 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 later found to be a new type of EHV, EHV-9, although it was serologically cross-reactive with EHV-1 (3). Recently, neutralizing antibodies against EHV-9 were found among Burchell’s zebras in the Serengeti ecosystem (4).

A herpesvirus was recently isolated from a reticulated giraffe (Giraffa camelopardalis reticulata) 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 seropositive for EHV-1 were housed in the same pen as the giraffe. The isolated virus was identified by PCR and a monoclonal 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 subunit (open reading frame [ORF] 30) gene was amplified by using herpesvirus 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 ORF33 primers were gB-F (5′-GGCCAAATAGTCCCAGCATG TCTGTTGCGT-3′) and gD-R (5′-AAATATCCTACGGGCGGAC TGGGAAAGTG-3′). The ORF71 primers were gp2-F (5′-CCCCGTGTAGTGTTTGCCTAGGCTCTA-3′) and gp2-R (5′-GCCACCCTAGGTTGTAAGGGCAAGGGTAT-3′). The ORF72 primers were gD-F (5′-TTTACACACGCTGGCGCT GTGTGCGAGAAC-3′) and gD-R (5′-TTATCTCAACCGGAGCTT TAAGGGCGGT-3′). The amplified products were used as templates for direct sequencing (Dragon Genomics, Mie, Japan). The sequences were edited with Phred, Phrap, and Consed (www.phrap.org/phredphrapconsed.html), and the phylogenetic trees were constructed with PHYLYP (2,7). Accession 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% identical 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 herpesvirus were 99.8% and 99.6% identical to EHV-9 and 91.6% and 96.3% identical to EHV-1. A phylogenetic tree of maximum likelihood showed that EHV-9 and the giraffe virus formed a genetic group that was apparently distinguished from other genetic groups of EHV (Figure).

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