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Streptobacillus moniliformis Endocarditis

To the Editor: *Streptobacillus moniliformis* is a facultatively anaerobic, pleomorphic, gram-variable bacillus often seen in chains and as long unbranched filaments. It is found in the nasopharynx and oropharynx of wild and laboratory rats. Human infections result either from rodent bites (rat bite fever) or contaminated milk or other foods (Haverhill fever). The most common manifestations of infection are arthralgia, fever, and rash; endocarditis occurs as a rare complication (*1*). We report a case of *S. moniliformis* endocarditis in India in a patient with congenital heart disease.

An 18-year-old man was admitted to the Department of Cardiology at the Government General Hospital in Chennai, India, in November 2005, with a fever of 2 months' duration with cough, epistaxis, palpitations, and persistent joint pain. His medical history indicated congenital heart disease with a ventricular septal defect. On physical examination, his blood pressure was 100/70 mm Hg, pulse rate was 100 beats/min, and temperature was 38.5°C. Laboratory tests showed a leukocyte count of 7,600/µL, a platelet count of 127,000/µL, and an erythrocyte sedimentation rate of 70 mm/h. An electrocardiogram showed normal sinus rhythm. A transthoracic echocardiogram demonstrated a ventricular septal defect and vegetations on the septal leaflet of the tricuspid valve.

Three blood cultures were prepared, and treatment with antimicrobial drugs (intravenous penicillin G, 3×10^6 U every 6 h, and gentamicin, 50 mg every 8 h for 4 weeks) was initiated. The blood cultures were incubated at 37°C in an atmosphere of 5%–10% CO_2. Characteristic white, downy, crumblike granules were observed on the surface of the erythrocytes in all 3 cultures within 18–24 h of incubation. Characteristic puff balls were seen after 48 h of incubation. Gram-stained smears showed gram-negative bacilli in long chains. Cultures were subcultured onto 5% sheep blood agar plates and MacConkey agar plates. The plates were incubated at 37°C in an atmosphere of 5%–10% CO_2. After 18–24 h of incubation, growth was seen on the sheep blood agar plates. Colonies were 1–2 mm in diameter, gray, smooth, and butyrous. A Gram stain of these colonies identified gram-variable, pleomorphic coccobacilli that were negative for catalase, oxidase, urease, and citrate, and did not produce indole or reduce nitrate.

Antimicrobial susceptibility testing was performed by using the Kirby-Bauer disk diffusion method according to recommendations of the National Committee for Clinical laboratory Standards (*2*). The isolate was sensitive to penicillin G, ceftriaxone, cephalexin, amoxicillin, gentamicin, and erythromycin. The patient responded well to treatment and became afebrile within 48 h after initiation of therapy. Treatment with antimicrobial drugs was continued for 4 weeks. The blood cultures were negative when repeated after 2 weeks. The patient had an uneventful recovery and was discharged from the hospital.

Rat bite fever is a zoonosis caused by either *Streptobacillus moniliformis* or *Spirillum minus* (*1,3*). *S. moniliformis* is found in the nasopharynx of small rodents, especially rats. Rats that are carriers have no symptoms but can effectively transmit the infection by bite or through infected body fluids such as urine.

This patient had a history of living in a rat-infested area, and admitted having been bitten by a rat several months before the onset of symptoms. However, we considered it unlikely that disease contracted by a rat bite would take months to be manifested. Thus, it is more likely that he contracted the infection from food or water contaminated with rat excreta. Endocarditis is a rare complication of *S. moniliformis* infection, and cardiac valvular abnormalities have been reported in 50% of cases (*4*). This patient, however, had only a small ventricular septal defect. This is the first report of *S. moniliformis* endocarditis from India.

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West Nile Virus in Horses, Guatemala

To the Editor: West Nile virus (WNV, *Flaviviridae*: *Flavivirus*) is emerging as a public health and veterinary concern. Since its introduction into North America in 1999, it has spread rapidly, reaching the Caribbean Basin in 2001, Mexico in 2002, El Salvador in 2003, and Colombia in 2004 (*1*). However, reports of equine illness and deaths in Latin America are inconclusive. With the exception of viral isolates from a dead bird, a human, and a mosquito pool in Mexico (*2,3*), all reports of WNV presence in Latin America have relied on serologic evidence. WNV is a member of the Japanese encephalitis serocomplex, which in the Western Hemisphere includes St. Louis encephalitis virus (SLEV) (*4*). Serologic investigations for WNV in Latin America must use highly specific assays to differentiate WNV infection from potentially cross-reactive viruses such as SLEV or possibly additional unknown viruses. In particular, SLEV is of concern since it was previously isolated from Guatemalan mosquitoes (*5*).

Alerted by the findings of WNV transmission in the region (*1*), we collected serum samples from horses from 19 departments of Guatemala from September 2003 to March 2004, to initially estimate the extent of WNV spread and its potential public health risk. Because no animals exhibited signs of neurologic illness at the time of the survey, only healthy horses were sampled. Before 2005, equine WNV vaccines were prohibited and unavailable in Guatemala (Unidad de Normas y Regulaciones, Ministerio de Agricultura Ganadería y Alimentación, Guatemala, pers. comm.); as such, cross-reactivity due to prior vaccination is highly unlikely. Samples were initially tested for WNV-reactive antibodies by using an epitope-blocking enzyme-linked immunosorbent assay (blocking ELISA) (*6*). The ability of the test sera to block the binding of the monoclonal antibodies to WNV antigen was compared to the blocking ability of control horse serum without antibody to WNV. Data were expressed as relative percentages and inhibition values ≥30% were considered to indicate the presence of viral antibodies.

A subset of positive samples was further confirmed by plaque-reduction neutralization test (*7*). Of 352 samples, 149 (42.3%) tested positive with the 3.1112G WNV-specific monoclonal antibody. Of 70 blocking ELISA–positive samples, the neutralization tests indicated the infecting agent was WNV, SLEV, and undifferentiated flavivirus in 9, 33, and 21 samples, respectively. Titers were expressed as the reciprocal of serum dilutions yielding ≥90% reduction in the number of plaques in a plaque-reduction neutralization test ($PRNT_{90}$). $PRNT_{90}$ titers of horses seropositive for WNV ranged from 80 to 320. $PRNT_{90}$ titers of horses seropositive for SLEV ranged from 40 to 2,560. For the differential diagnosis of samples with neutralizing antibody titers against both WNV and SLEV in this test, a ≥4-fold titer difference was used to identify the etiologic agent. The undifferentiated flavivirus-reactive specimens had <4-fold difference in cross-neutralization titers. Likely possibilities for the inability to distinguish the infecting virus include previous infection with these or other flaviviruses (previously described or unknown) resulting in elevated cross-reactive titers. The remaining 10% of specimens that tested negative by PRNT probably represent nonneutralizing antibodies in the serum or false positivity in the blocking ELISA.

Our serologic results provide indirect evidence of past transmission of WNV, SLEV, and possibly other flaviviruses to horses in Guatemala. Although no confirmed cases of WNV-attributed disease have been reported in Central America to date, flavivirus transmission appears to be widely distributed in Guatemala (Figure). Efforts are under way to confirm WNV transmission by viral isolation and to evaluate the impact of WNV on human, horse, and wildlife populations. More information is needed to establish the public health threat of WNV and other zoonotic flaviviruses in the region.

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