

# Unprotected People #84

## Rabies

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### Mississippi boy dies from rabies; exposure to bats around home is likely source

*On September 27, 2005, a previously healthy 10-year-old Mississippi boy died from encephalitis later attributed to rabies. Following his death, this case was referred to CDC's Unexplained Deaths Project. Serum and cerebrospinal fluid collected from the boy when he was living led to a diagnosis of rabies. This was the only case of human rabies diagnosed in the United States in 2005.*

*The presence of bats in and around the boy's home was the likely source of the boy's exposure to rabies; in addition, it was reported that the boy had handled a live bat in spring 2005. Timely prophylaxis after bite, scratch, or mucous-membrane exposure to wildlife can prevent rabies. Once clinical signs of rabies develop, postexposure prophylaxis is no longer effective. This report underscores the need for increasing public awareness of the risk of rabies after contact with bats and other wildlife.*

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#### **Human Rabies—Mississippi, 2005**

On September 27, 2005, a previously healthy boy aged 10 years in Mississippi died from encephalitis later attributed to rabies. This report summarizes the patient's clinical course and the subsequent epidemiologic investigation, which implicated exposure to bats at the boy's home as the likely

source of rabies. The findings underscore the importance of recognizing the risk for rabies from direct contact with bats and seeking prompt medical attention when exposure occurs.

#### **Case Report**

On September 11, 2005, the boy had fever and headache. He was evaluated by a pediatrician on September 13 for a temperature of 102.4°F (39.1°C) and was noted to have sensations that the patient described as an "itchy" scalp. Viral illness was diagnosed, and the patient was advised to return if symptoms worsened. The patient was taken to a local emergency department (ED) in the early morning hours of September 15 with ongoing febrile illness. All laboratory tests and chest radiography ordered were within normal limits, and the patient was discharged home.

The patient's clinical signs worsened throughout the day, and he returned to the ED that evening with symptoms of fever, insomnia, urinary urgency, paresthesia of the right side of the scalp and right arm, dysphagia, disorientation, and ataxia. He was admitted to the hospital for suspected encephalitis. A clinical history revealed no known tick bites or contact with animals other than the family pets.

Upon admission, the patient had a temperature of 100.0°F (37.8°C) and a white blood cell (WBC) count of 12,200/microliter (normal: 4,800–13,500/microliter). Analysis of cerebrospinal fluid (CSF) indicated a WBC count of 226/microliter (normal: 0–5/microliter), protein level of 79 mg/dL (normal: 12–60 mg/dL), and glucose level of 69 mg/dL (normal: 45–75 mg/dL). Serum and CSF samples were obtained for IgG and IgM antibody testing for

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West Nile, St. Louis, Lacrosse, and Eastern equine encephalitis (EEE) viruses.

Shortly after admission, the patient's neurologic status deteriorated rapidly. His speech became slurred, and he began to hallucinate. He became increasingly agitated and combative and required sedation. In his agitated state, the patient bit a family member. The next morning the patient was transferred to a tertiary care facility. Within hours after transfer, he became lethargic and was intubated. Serologic tests for West Nile, St. Louis, Lacrosse, and EEE viruses, Rocky Mountain spotted fever, and Bartonella spp. were negative. Herpes simplex virus and enterovirus were not detected in CSF by polymerase chain reaction (PCR), and arbovirus-specific antibodies were not detected in CSF. Computed tomography scans of the head with and without contrast were within normal limits. During the next 10 days, the patient continued to worsen and experienced wide fluctuations in blood pressure and temperature. On September 26, he had onset of cerebral edema and subsequent brain herniation. Life support was withdrawn, and the patient died on September 27.

### **Laboratory and Public Health Investigation**

The case was referred to CDC's Unexplained Deaths Project (UNEX) for additional diagnostic testing. Clinicians who had treated the patient suspected EEE and possibly rabies on the basis of the patient's rapidly progressive encephalopathy. On October 5, CDC diagnosed rabies on the basis of an increase in rabies-virus-specific IgG antibody titer from 128 to 8,192 in paired sera samples collected on September 16 and 21. Subsequent testing of CSF demonstrated the presence of rabies-virus-specific antibodies. Rabies-virus nucleic acid was not detected in CSF by reverse transcription PCR. No other clinical specimens were available to allow virus characterization and identification of a likely animal source of infection.

Family members and friends of the patient did not report a definitive animal bite when queried during the patient's illness. However, after the child's death, several persons reported that bats were commonly seen outside the home. On two occasions, dead

bats also were discovered inside the home and attached garage, and a live bat was caught in an apartment above the garage during the summer of 2005. The child had removed a live bat from his bedroom and released it outdoors in the spring of 2005.

The child had attended a summer camp in Alabama for several weeks in July. The camp program included an overnight stay in a nearby cavern used for tours and special events. Interviews with the camp director and parents of children who attended the overnight camp-out with the patient revealed no indication of direct contact with bats at the camp or in the cavern, although one bat was reportedly observed clinging to the rocky wall inside the cavern.

Postexposure prophylaxis (PEP) was administered to 23 family members and friends who possibly had contact with the patient's saliva from August 28 (14 days preceding the first clinical signs of rabies) to the patient's death on September 27. Interviews with family and friends suggested that the patient commonly shared food and drink with others, particularly children. Among 79 healthcare workers evaluated for potential exposure to infectious body fluids, 32 received PEP, including 19 nurses, four physicians, five respiratory therapists, two radiology technicians, and two laboratory staff.

### **Editorial Note**

This report describes the only case of human rabies diagnosed in the United States in 2005 and the first case in Mississippi since 1956. On the basis of multiple reports regarding the presence of bats in and around the family home in Mississippi and the observation that the patient had handled a live bat at his home in the spring of 2005, contact with a bat at the patient's home was determined to be the likely source of rabies infection in this case. Bats are the only known reservoir of rabies in Mississippi.

Since 1995, a total of 379 deaths possibly attributed to infectious disease have been reported to CDC's UNEX. Of these, 131 (35%) have had a probable etiology identified. The case described in this report

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represents the first diagnosis of rabies made for a death reported to UNEX.

Thirty-two healthcare workers received PEP as a result of this case. Providing health care to a patient with rabies is not an indication for PEP unless mucous membranes or an open wound are contaminated with infectious material, such as saliva, tears, CSF, or neurologic tissue. Standard precautions and adherence to infection-control measures will minimize the risk for exposure.

During 1980–2004, a total of 56 cases of human rabies were reported in the United States. Among the 55 cases for which rabies-virus variants were obtained, 35 (64%) were associated with insectivorous bats, most commonly the silver-haired and eastern pipistrelle bats. More than half (57%) of these human cases occurred during August–November, coincident with a seasonal increase in prevalence of rabid bats detected in the United States. Despite the substantial number of cases of human rabies attributable to bat exposure, the importance of these exposures is often overlooked or under-estimated.

Human rabies is preventable with proper wound care and timely and appropriate administration of PEP after exposure. PEP is recommended for all persons with a bite, scratch, or mucous-membrane exposure to a bat unless the bat tests negative for rabies. When a bat is found in close proximity to

humans, it should be submitted to a public health laboratory for diagnostic testing, if it can be captured safely. If the animal is not available for testing, PEP should be administered when a strong probability exists that exposure occurred. However, if a bat bite is unrecognized or the importance of the exposure is underestimated, medical intervention might not be sought and appropriate treatment might not be administered. Once clinical signs of rabies develop, PEP is no longer effective and a rapid, progressive, and usually fatal encephalitis ensues.

This report underscores the need for increasing public awareness of the risk for rabies after contact with bats and other wildlife. Persons bitten by a potentially rabid animal should immediately (1) wash the wound thoroughly with soap and water; (2) capture the animal, if this can be done safely (avoiding direct contact with the animal) and submit it for testing; (3) report the incident to local or state public health officials; and (4) see a physician for treatment and evaluation regarding the need for PEP. Persons should not handle or keep bats as pets and should exclude bats from living quarters, public places, and structures adjacent to the home. Recognizing the risk for rabies from any direct exposure to bats and other wildlife is critical, and persons must seek prompt medical evaluation if exposed.