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HUMAN RABIES DIAGNOSED 2 MONTHS POSTMORTEM -- TEXAS

The first case of human rabies reported in the United States in 1985 was diagnosed July 16, 1985, by an Abilene, Texas, pathologist who noted encephalitis suggestive of rabies on reviewing sections of the brain of a patient who had died May 20. The patient, a 19-year-old Mexican national, had lived in Texas after arriving in the United States approximately 1 1/2 months before the onset of his illness. He had no known history of exposure to rabies.

The patient was in good health until May 2 or 3, when he developed nausea, vomiting, and shortness of breath. On the morning of May 5, he was seen at the emergency room of an Abilene hospital. Temperature, pulse, and blood pressure were normal. Physical examination and a chest roentgenogram did not reveal abnormalities, and the patient was discharged from the emergency room.

Shortly after midnight on May 6, he returned to the emergency room because of intensification of breathing difficulties, persistent nausea and vomiting, and fever of 40.6°C (105°F). His blood pressure fluctuated between 215/140 and 80/0. He was coherent enough to answer questions in Spanish; however, because he spoke no English, no detailed history of his activities for the past several months was obtained. Tetanus and rabies were considered, but both were ruled out because of a negative history of an injury or animal bite. Admission white blood cell count (WBC) was 25,800/mm^3 and hematocrit, 49%. An arterial blood gas sample revealed a metabolic acidosis. Serum potassium was 2.9 meq/l; glucose, 389 mg/dl;lactate, 12.2 meq/l; and serum acetone, negative. Urinalysis showed a trace of protein, mild ketonuria, and 3+ glucose. He was admitted to the hospital's coronary-care unit in acute respiratory distress with a provisional diagnosis of sepsis and rupture of the esophagus, but a cine-esophagram did not confirm the latter diagnosis. A repeat chest roentgenogram examination showed air in the neck and mediastinum and right-lung infiltrates. Aspiration pneumonia was suspected. The patient was intubated for respiratory distress approximately four hours after admission and was treated with broad-spectrum antibiotics. Blood and stool cultures for bacteria and a blood smear for malaria parasites were negative. A drug screen of serum showed only a positive reaction for acetaminophen.

The patient improved enough by May 8 to have the endotracheal tube removed. However, over the next day, his neurologic condition deteriorated, and he became disoriented and combative. Tremors were noted in his neck. A neurology consultant felt the patient's disorientation was metabolic in origin, but suggested cerebrospinal fluid examination. The initial lumbar puncture, performed May 12, showed 3 RBCs/mm^3, 14 WBCs/mm^3 (86% lymphocytes and 14% neutrophils) and 159 mg/dl of protein. On May 13, the patient suffered respiratory arrest and required reintubation. Over the next seven days, his course was marked by progressively deepening coma without focal signs. His electroencephalogram showed a slow-wave pattern. The patient died May 20, two weeks after admission.

Since rabies was not seriously suspected during the patient's illness or at autopsy, microscope examinations of the brain and other tissue specimens were given routine rather than expeditious scheduling. Consequently, microscope examination of the

brain was not undertaken until early July, when the pathologist reviewed the sections from the brain. The histologic diagnosis was further supported on July 16 by a Houston neuropathologist. On July 18, formalin-fixed brain tissue preserved from the autopsy was forwarded to CDC for examination. Direct fluorescent-antibody examination gave strongly positive results, and rabies was confirmed.

On July 19, local, regional, and state public health physicians met the members of the medical staff, hospital administrators, and approximately 140 hospital employees who had had contact with the patient. Rabies postexposure prophylaxis was made available to the employees and staff members by the hospital; 85 workers elected to take the treatment. Postexposure treatment was also offered to relatives and friends who could be located; they denied exposure to the patient's saliva or vomitus and chose to receive no treatment. Cost of rabies immune globulin and human diploid cell rabies vaccine was approximately $29,000.

[This report was prepared by C. E. Alexander, MD, DrPH, Chief: Bureau of Epidemiology, and Tracy L. Gustafson, MD, Director: Infectious Disease Division, Texas Department of Health.]

Editorial Note: Of the 47 rabies cases diagnosed in the United States (or in American citizens outside the United States) and reported to CDC since 1960, no history of exposure could be ascertained for 13 (28%). A median incubation period of 35 days (range 12-701) was determined for the other 34 cases. In the present case, the absence of a history of a bite or other contact with a possibly rabid animal may have been attributable to memory loss resulting from encephalitis or to miscommunication because of the language barrier. Although the source of exposure is unknown, the patient's 1 1/2-month residency in the United States is compatible with exposure in Texas or Mexico. In the semiarid plains of Texas, skunks are the principal reservoir for rabies, although rabid bats and foxes play an occasional role in the transmission of the infection in that region. In Mexico, dogs account for most reported cases of rabies.

Five (56%) of the nine rabies cases reported to CDC since 1980 occurred among individuals who had recently lived in rabies-endemic areas outside the United States. The last two cases were foreign nationals who developed rabies shortly after arrival in the United States from rabies-endemic areas.[1] In both, rabies was diagnosed postmortem.

When encephalitis occurs in a person who has lived in an area where rabies is enzootic, the diagnosis should be considered seriously, even in the absence of a history of exposure. Suggestive of rabies in the present case, in addition to encephalitis, were agitation, progressive unexplained dysphagia, and later in the course of illness, fasciculations of the neck.

Although the prognosis for recovery after onset of clinical illness is bleak, early suspicion of rabies will allow for rapid institution of isolation measures to reduce the number of persons exposed to the patient and eliminate most exposures that might occur in situations such as airway care, provision of oral and dental hygiene, and physical examination of the head and neck.[2]

The low risk of rabies transmission to hospital personnel caring for a rabid patient[3] is supported by the absence of rabies cases in hospital contacts of the patient despite a 60- to 78-day delay in instituting postexposure prophylaxis. Postexposure prophylaxis is recommended after contact with a rabid human only if a bite or nonbite exposure (contamination of a mucous membrane or open wound with saliva or other potentially infectious material) occurred.[2,4] When only persons known to be exposed are treated, unnecessary postexposure treatments can be discouraged, and substantial savings can result. Consultation with state or federal health officials experienced in evaluating human rabies is recommended.

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[Also published in: CDC. MMWR;34:700,/05-70/.]

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HYSTERIA OUTBREAK IN WEST TEXAS

On Monday, September 30, 1985, the Bureau of Epidemiology was notified of a possible gastroenteritis outbreak involving high school students who attended an out-of-town football game on Friday, September 27. Thirty students, all members of the high school marching band, arrived at the local hospital emergency room on Friday night, complaining of abdominal cramps and muscle spasms. The duration of illness was one to two hours. No fever, vomiting, or diarrhea was noted. Twenty-nine of the ill students were females.

The students had eaten at a local restaurant between 6PM and 7PM Friday. A total of 117 individuals from their high school ate the meal which consisted of chicken fried steak, gravy, rolls, green salad, french fried potatoes, banana pudding, and tea.

The epidemiologic and clinical characteristics of this outbreak are suggestive of epidemic hysteria. Hysteria outbreaks are characterized by: 1) a preponderance of female cases, 2) clustering of cases by time and location, 3) rapid onset and remission of symptoms, 4) the lack of physical findings despite subjective complaints, and 5) an adolescent age group affected in connection with a group event.[1-3] Most outbreaks of hysteria have occurred in schools or have involved children. Possible control measures could include: 1) dispersion of the affected group, 2) isolation of the affected group from others, and 3) an early announcement of the nature of the outbreak.

This report was prepared by Jeffery P. Taylor, MPH, Staff Epidemiologist, Infectious Disease Division, TDH.

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CHROMOSOMALLY-MEDIATED-RESISTANT NEISSERIA GONORRHOEAE (CMRNG)

Since August 1985, there have been 18 CMRNG cases reported to the Venereal Disease Control Division. These cases are non-beta-lactamase producing but are penicillin resistant.

Epidemiological work-up of these cases indicates the source infection to be indigenous to the area of patient residence. Most of the CMRNG cases have been reported from the metropolitan areas of San Antonio (7) and Dallas (5) and the lower Rio Grande Valley (Harlingen-3 and McAllen-1).

Health providers performing gonorrhea cultures are encouraged to screen all positive test-of-cure specimens and all negative beta-lactamase specimens for CMRNG. Screening by disk agar diffusion or with penicillin-containing media will identify chromosomally mediated resistance to penicillin.

Recommended treatment for CMRNG is spectinomycin 2.0 g IM or ceftriaxone 250 mg IM.

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NOTE

Addendum: "Immunization Clinic Delinquent Systems," TPDN, Vol. 46, No. 2. Clinic Delinquent Cards (Form C-64) are provided by both the Texas Medical Association and the Texas Osteopathic Medical Association.

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