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Texas Preventable Disease NEWS

contents:

BUREAU OF EPIDEMIOLOGY 1100 West 49th Street, Austin, Texas 78756 (512-458-7207)

ARBOVIRAL INFECTIONS IN TEXAS

The isolation of western equine encephalitis (WEE) virus from mosquitoes in Liberty County, PHR 11, and demonstration of antibodies to eastern equine encephalitis (EEE) virus in sentinel chicken flocks in Galveston County marks the beginning of the arboviral season in Texas for 1985. Physicians throughout the state are reminded to consider arboviral infections in patients presenting with symptoms of acute central nervous system (CNS) infections.

Several arboviruses are endemic in Texas. St. Louis encephalitis (SLE) virus and WEE virus have caused major outbreaks in the past. The most recent outbreaks of illness due to SLE virus were in 1980, when there were 68 cases, primarily in the Houston area, and in 1982, when there were 18 cases, again in the Gulf Coast area. Although isolations of WEE have been made in various parts of the state, cases have been reported only from western Texas in the past decade. The last cluster of cases was in 1982, when four cases were reported. Sporadic infections of both viruses have also occurred in other years.

Arboviruses produce CNS infections that range from mild febrile illnesses to aseptic meningitis and encephalitis. SLE infections are generally more severe in older patients. However, of the four cases reported in 1981, the two adults recovered without sequelae, but the two children, both under 10 years of age, had severe illness with long-term sequelae involving mental retardation and permanent loss of motor coordination. In contrast, WEE infections are typically more severe in children, particularly infants. In this group, the infection may be characterized by unremitting seizures which are not responsive to therapeutic measures. EEE virus has been demonstrated in eastern Texas but has rarely caused illness, since the vector, Culiseta melanura, does not readily feed on humans. However, when illness does occur, the mortality rate is significant, approximately 50%.

Dengue is another arbovirus that has caused illness in Texas in the past. In 1980, indigenous cases occurred for the first time in 26 years. These cases were part of an outbreak that spread from the Caribbean islands to the continent, then north through Mexico to Texas. Infections with this virus produce a severe influenza-like disease; CNS manifestations are rare. Only three cases of dengue have been reported since 1980; all were in Texas residents who had acquired their infections while traveling outside the United States

Viruses of the California encephalitis group have been isolated in Texas, but none of these species has been associated with disease. However, in other parts of the United States, one member of this group, LaCrosse encephalitis virus, is the most common cause of summertime encephalitis, particularly among children.

Although the other arboviruses discussed are endemic in certain parts of Texas--SLE along the Gulf Coast, WEE in western Texas, and EEE in the eastern counties--cases

are not always diagnosed in those limited areas. For example, the first case of SLE reported during the 1980 outbreak was diagnosed in Wichita Falls. This individual had been vacationing on the Gulf Coast two weeks prior to onset. Physicians throughout Texas should consider arboviral infection in the differential diagnosis for patients with compatible symptoms and elicit a travel history for the two weeks prior to the onset of illness. The incubation period for all of these arboviral infections ranges from ten to 14 days.

Arboviral infections are confirmed serologically by demonstration of a four-fold rise in antibody titer between acute and convalescent sera collected at least two weeks apart. Such testing is available only at the Texas Department of Health in Austin and the Houston City Health Department. All sera submitted for arboviral testing are tested against the entire battery of arboviral agents.

Enteroviruses, another common cause of summertime CNS infections, give rise to the same spectrum of symptoms as arboviruses, ranging from a mild febrile illness through aseptic meningitis to encephalitis. Enteroviral infections, unlike arboviral infections, are diagnosed by demonstration of the virus in cerebrospinal fluid (CSF), stool, or nasopharyngeal secretions rather than by serology. Specimens to be submitted for viral isolation must be sent cold to the TDH Bureau of Laboratories in Austin.

Although confirmation of a diagnosis may take a period of weeks to months, control measures must begin as soon as an arboviral infection is suspected. Control of arboviral diseases requires close cooperation between the public and private health care sectors. Physicians who suspect arboviral infections in their patients should report such cases, even prior to confirmation, to their local health authorities so that appropriate mosquito control measures can be initiated quickly. Control measures generally involve: 1) a visual assessment of the patient's physical environment (eg, home, work, and recreational sites) for the possible presence of vector mosquitoes, and 2) appropriate vector eradication efforts. In many areas where arboviral infections have occurred in the past, mosquito control measures are undertaken routinely. Upon notification of a suspect case in a community, these measures may simply be redirected to a different area of town.

Questions concerning mosquito control measures should be directed to the TDH General Sanitation Division at (512) 458-7521 in Austin. The Bureau of Laboratories, Medical Serology Branch may be reached at (512) 458-7514 and the Medical Virology Branch, at (512) 458-7515. General questions may be directed to the Bureau of Epidemiology at (512) 458-7328.

This report was submitted by Christie Reed, MPH, Staff Epidemiologist, Bureau of Epidemiology, Texas Department of Health.

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NOTICE TO READERS

The Editor of Texas Preventable Disease News (PDN) welcomes written accounts of communicable disease and other public health problems encountered and investigated by local health professionals throughout the state. During 1982, numerous articles published in PDN were contributed by individual health care workers in Texas. The Bureau of Epidemiology encourages public health workers to share their experiences and information relating to matters of professional public health interest or concern. Previously published accounts of this nature have been favorably received by the readership. Interested authors are requested to contact the Editor of PDN for additional information pertaining to general guidelines for publication at (512) 458-7207 or Tex-An 824-9208.

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FATAL DEGENERATIVE NEUROLOGIC DISEASE IN PATIENTS WHO RECEIVED PITUITARY-DERIVED HUMAN GROWTH HORMONE

This article first appeared in the Centers for Disease Control (CDC) publication, Morbidity and Mortality Weekly Report, Vol. 34/No. 24, June 21, 1985.

Reports of rapidly progressive and fatal degenerative neurologic disorders in three recipients of human growth hormone (hGH) have been received by the US Food and Drug Administration (FDA) and the National Institutes of Health (NIH). In two cases, diagnoses of Creutzfeldt-Jakob disease (CJD) were made at autopsy.

All three patients had had growth failure secondary to growth hormone deficiency. They had been treated during childhood and adolescence with hGH extracted from pooled human cadaver pituitary glands. The hormone used to treat these patients was produced and distributed by the National Hormone and Pituitary Program (NHPP, formerly the National Pituitary Agency) under an investigational exemption for the use of a new drug(IND).

Case 1. A 20-year-old man with hypopituitarism and Type I diabetes mellitus developed dysarthria and a gait disturbance in May 1984. By September, his neurologic status had deteriorated so that he was no longer able to walk, could not care for himself, and required bladder catheterization. His mental status had deteriorated, and he was unable to carry on a meaningful conversation. He died in November 1984. Examination of the brain revealed spongiform encephalopathy consistent with CJD.

This patient had grown poorly during the first year of life. Hypothyroidism was diagnosed when he was 15 months old. In September 1966, a diagnosis of growth hormone deficiency was made. The patient was treated with daily injections of hGH from September 1966 to July 1980.

Case 2. A 22-year-old man developed weakness and gait disturbance in the fall of 1983. During the next 6 months, he developed severe ataxia involving extremities, trunk, and head. He also had speech impairment, difficulty swallowing, and dementia. He died in April 1985. Histologic examination of the brain at the Armed Forces Institute of Pathology revealed extensive changes of spongiform encephalopathy compatible with CJD.

This patient was evaluated for growth failure at 7 years of age and was found to be growth hormone deficient. He was treated with hGH from June 1969 through October 1977.

Case 3. A 34-year-old man with hypopituitarism developed a gait disturbance in December 1983. He had received hGH from 1963 to 1969. Examination in June 1984 showed bilateral horizontal end gaze nystagmus, mild intention tremor, and wide-based gait. The symptoms worsened over the next several months, with increasing somnolence, memory loss, and urinary incontinence.

The patient's symptoms progressed to include swallowing difficulties, diplopia, and finally, dementia. He died in February 1985. No autopsy was done.

MMWR Editorial Note: CJD occurs with a frequency of approximately one case per million population per year in the United States and Europe. Most cases occur sporadically and involve patients over 50 years of age. Innoculation of chimpanzees with brain tissue from affected patients results in a similar neurodegenerative disease in the animals within 18 to 36 months. Iatrogenic CJD has been reported in a

patient who received a corneal transplant from an affected donor and in two patients exposed to intracranial electrodes that had previously been used in a patient with CJD.

The CJD pathogen is resistant to chemical and physical methods commonly used for decontamination or sterilization. There is evidence suggesting that procedures used recently to extract and purify hGH from cadaver pituitary glands may eliminate experimental contamination by scrapie, an agent similar to the CJD pathogen. The methods used by the NHPP were changed in 1977, but there is no assurance that current procedures eliminate the risk of transmitting the CJD pathogen.

From 1963 to early 1985, approximately 10,000 US patients received hGH through the NHPP. The average duration of therapy was 4 years. Each patient received hormone from two or three batches per year. Each batch was derived from a pool of approximately 16,000 cadaver pituitary glands.

The three patients described here received hGH for 14, 8, and 6 years, respectively. Records of the NHPP indicate that patients 1 and 2 received several common lots. Patients 1 and 3 received one lot in common. No single lot was administered to all three patients; however, all three received hormone during 1969. The occurrence of fatal neurodegenerative disorders consistent with CJD in three of 10,000 patients exposed to hGH between 1963 and 1985 strongly suggests that the hormone, a product of pooled human tissue, may have been the vehicle for transmission of the CJD pathogen. It is not yet known how many other members of this cohort may have developed similar neurodegenerative disorders. Epidemiologic studies will be undertaken to determine the status of recipients of hGH.

Patients under 40 years of age with progressive dementing neurodegenerative disorders who may have received pituitary derived human growth hormone either through the NHPP or from commercial sources should be reported to: E Rappaport, MD, HFN-810, Center for Drugs and Biologics, US Food and Drug Administration, 5600 Fishers Lane, Rockville, Maryland 20857; telephone (301)443-3520.

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