Tropical Storm Allison Rapid Needs Assessment— Houston, Texas, June 2001

ON JUNE 5, 2001, TROPICAL STORM ALLISON made landfall on Galveston Island, Texas. During the next 2 days, the system soaked much of southeast Texas and south-central Louisiana with more than 10 inches of rain as it moved slowly northward. On June 7, the storm made a clockwise loop back to the southwest, bringing even more rain to already drenched areas. The record rainfall caused billions of dollars in flood-related damage and approximately 25 deaths and led to a presidential disaster declaration covering 31 Texas counties and 28 Louisiana parishes. Harris County, Texas (2000 population: 3,400,578), center of the Houston metropolitan area, was among the hardest hit with some areas receiving up to 37 inches of rain in 24 hours. To evaluate the community’s immediate public health needs, the City of Houston Department of Health and Human Services (HDHHS) conducted a rapid needs assessment in the areas most affected by flooding. This report summarizes assessment results, which identified increased illness in persons living in flooded homes, suggesting a need for rapid resolution of flood-related damage and the possibility that residents should seek temporary housing during clean-up and repair. The findings underscore the usefulness of rapid needs assessment as a tool to minimize misinformation, identify actual health threats, and ensure delivery of resources to those with the greatest and most immediate need.

Severely flood-affected areas of Houston, identified by the City of Houston Office of Emergency Management, were divided into two sectors: Area A, in the northeast section of the city, comprised 96 census tracts, 148,654 housing units, and 443,356 residents; and Area B, covering the western part of the city, comprised 72 census tracts, 167,158 housing units, and 400,868 residents. Using modified cluster sampling, HDHHS obtained a random sample of 30 census tracts from each of the two areas by using statistical software (SPSS V.10). One neighborhood from each of these 60 tracts was selected by delineating block groups using 1997 household estimates (housing unit data from the 2000 census were not yet available), breaking into quintiles the distribution of household population, and selecting the block group with the highest population and concentration of streets. A standardized questionnaire was developed to collect information about demographics, extent of home damage, number of residents forced to leave their homes, reported illnesses and injuries among household members, disruption of public services, and current needs. The goal was to obtain completed questionnaires from seven households in each of the 60 selected census tracts for a total sample of 420 households. Data were analyzed by using SAS for Windows (release 8.01). Data were stratified by census tract for calculation of illness and injury odds ratios for living in a flooded versus nonflooded home.

HDHHS conducted the survey door to door on June 16, 2001, 1 week after the heaviest rainfall caused the worst flooding, and met its goal of 420 completed questionnaires. The overall response rate was 59.3%; nonresponses included 257 instances of no one at home, 20 refusals, seven uninhabited households, and four households in which a language barrier prevented communication. Of the 420 households surveyed, 389 (92.6%) were single-family homes, and mean household size was 3.3 persons (range: 0-20 persons). A total of 137 (32.6%) surveyed households had floodwaters in the home; mean floodwater depth was 16 inches (median: 12 inches, range: 1-60 inches), and mean duration floodwaters remained in the home was 36.8 hours (median: 24 hours, range: 1-168 hours). A total of 149 (35.5%) surveyed households reported damage to the home; all but one of these were either habitable (116 [77.9%]) or repairable (32 [21.5%]). Survey participants in 57 (13.6%) households reported spending at least 1 night away from home. For some period during or after the flooding, 138 (32.9%) households reported interruption in telephone service, 63 (15.0%) had no sewage service, 61 (14.5%) lost electricity, 44 (10.5%) lost natural gas supply, and 23 (5.5%) had no running water. At the time of the survey, some households were still without telephone service (38 [9.0%]), natural gas (29 [6.9%]), sewage service (21 [5.0%]), electricity (eight [1.9%]), or running water (eight [1.9%]). The most commonly identified needs were mosquito control, pharmacy access, and new household furnishings. Other needs included medical access, shelter, food, home repair, clothing, transportation, drainage assistance, financial assistance, and heavy trash pick-up.

Fifty-four (12.9%) surveyed households reported at least one person with illness that occurred after the onset of flooding. Persons living in flooded homes were significantly more likely than those living in nonflooded homes to report illness; the only specific illness significantly associated with residing in a flooded home was diarrhea/stomach conditions. A total of 17 (4.0%) surveyed households reported at least one person injured after the onset of flooding. No significant association existed between likelihood of reporting an injury and living in a flooded home.
of illnesses and injuries reported by clinic-based surveillance conducted immediately post-disaster and those self-reported through a survey a week later. Furthermore, persons not at home and therefore unable to participate in the needs assessment survey might have been away from home because of illness or injury.

In addition to the potential for actual flood-related health impacts, rumors of epidemics often follow floods and other natural disasters and quickly gain public credibility when reported by the media. Actual threats and the potential for rumors combine to underscore the need for rapid information gathering to facilitate decision making and address public concerns. Rapid needs assessment—combining epidemiologic, anthropologic, and statistical methods—provides accurate information quickly and at low cost to minimize misinformation and identify actual health threats. This assessment was planned, conducted, and analyzed within 1 week and quickly provided important information to the City of Houston Mayor’s Office, City Council members, City of Houston Office of Emergency Management, American Red Cross, Federal Emergency Management Agency, CDC’s Emergency Response team, Texas Department of Health, and other agencies assisting with flood recovery efforts. These findings underscore the usefulness of rapid needs assessment as a practical and responsive data-gathering tool to complement clinic-based surveillance in disaster settings. The results were integral in assessing damage, setting priorities for service delivery, and directing assistance efforts. Numerous persons with special needs were identified and the appropriate assistance agencies quickly notified, heavy trash pick-up crews were redirected to neighborhoods where they were needed most, and residents of an area with chronic drainage problems were put in contact with the city engineering department to work out plans for future improvements. The results of this assessment will guide future needs assessments, disaster-response planning, and disaster-effects mitigation.

REFERENCES


Cat-Scratch Disease in Children—Texas, September 2000–August 2001

MMWR. 2002;51:212-214

Cat-scratch disease (CSD), a bacterial infection caused by Bartonella henselae, has emerged as a relatively common and occasionally serious zoonotic disease among children and adults. To illustrate the spectrum of clinical manifestations of CSD observed during a 1-year period, Texas Children’s Hospital (TCH) in Houston reviewed the medical records of 32 children evaluated at TCH during September 2000–August 2001 whose antibody titers indicated recent Bartonella infection. This report summarizes the evaluations of these cases and high-
lghts four manifestations of infection with this pathogen in children. The findings emphasize that although CSD is generally a mild, self-limited illness, the differential diagnosis often includes more serious conditions (e.g., lymphoma, carcinoma, mycobacterial or fungal infection, or neuroblastoma) that might result in protracted hospital stays and lengthy treatments before diagnosis. Timely assessment of CSD is important, particularly when invasive diagnostic measures are being considered.

Case Reports

Case 1. In July 2000, a boy aged 5 years was admitted to a local hospital after having fever (with temperature reaching 104°F [40°C]) for 12 days and left upper quadrant pain for 8 days. Aspartate and alanine aminotransferase concentrations were normal; a blood culture grew a contaminant. The child was transferred to TCH for evaluation of unexplained fever. Except for fever and inflamed tympanic membranes, the physical examination was unremarkable. Peripheric white blood cell count was 18.3 × 10^3/cu mm (normal range: 5-14.5 × 10^3/cu mm), erythrocyte sedimentation rate (ESR) was 97 mm/h (normal range: 0-20 mm/h), and IgG and IgM serologic test results for Epstein-Barr virus (EBV) were negative. Because the child had exposure to kittens and birds, doxycycline was administered along with penicillin, cefotaxime, and gentamicin at the time of transfer back to the referring hospital. The B. henselae titer obtained on day 7 at TCH was 1:8192.

Case 2. In September 2000, a girl aged 10 years with a bicommisural aortic valve had persistent low-grade fever, myalgias, arthralgias, weight loss, splinter hemorrhages, and hematuria and was admitted to TCH for evaluation and surgical management of endocarditis. She had been evaluated during the previous 9 months at another medical center for culture negative endocarditis. A transesophageal echocardiogram showed aneurysmal dilatation of the ascending aorta and probable vegetations. She also had a pulsatile lesion on the right forearm. Endocarditis caused by Chlamydia psittaci was suspected on the basis of the patient’s history of bird contact. During surgery, a large pseudoaneurysm of the ascending aorta and thickened dysplastic aortic valves were replaced with an aortic valve homograft. Histology demonstrated microabcess formation at the mouth of the aneurysm, noncaseating granulomatous inflammation in the wall of the aneurysm, and numerous granulocyte negative bacilli within vegetations. She also had resection of a brachial artery aneurysm with reconstruction of the artery. All cultures of tissue were sterile. Serologic test results for Coxiella burnetii, Brucella spp., Histoplasma capsulatum, and Coccidioides immitis were negative. Because the child had exposure to kittens and birds, doxycycline was administered along with penicillin, cefotaxime, and gentamicin at the time of transfer back to the referring hospital. The B. henselae titer obtained on day 7 at TCH was 1:8192.

Case 3. In June 2001, a boy aged 4 years was admitted to TCH with a 4-day history of intermittent back pain and an inability to walk. He had no history of trauma or contact with cats. He had a temperature of 99°F (36.6°C), no tenderness over the vertebrae, normal reflexes, and a 2x3 cm right inguinal lymph node. ESR was 93 mm/h. Two blood cultures and a urine culture were sterile. Stool cultures for various bacterial pathogens, including Yersinia enterocolitica, were negative. Several enlarged lymph nodes in the right lower quadrant were found on an ultrasound of the abdomen, but an abdominal CT was normal. Serologic test results for Toxoplasma gondii, cytomegalovirus, and EBV were negative. On day 7 of hospitalization, the patient underwent a colonoscopy, which was normal except for nodularity with mucosal edema in the terminal ileum. She had a recent history of dog and kitten scratches. Her B. henselae titer obtained during week 4 of illness was >1:8192.

Of the 32 patients, median age was 6 years (range: 2–15 years). Among the remaining 28 CSD cases observed at TCH during this 1-year period, clinical manifestations included fever and regional adenopathy (classic CSD) (20); prolonged fever without organ involvement (four); hepatosplenic granulomatata (three); and encephalitis (one). Fourteen of the children were hospitalized.

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CDC Editorial Note: CSD was first described as a clinical syndrome in 1931, but it was not until 1983 that a bacterial etiology was determined, and in 1992, the specific cause of CSD was identified. CSD is a feline-associated zoonotic disease, with an estimated annual incidence in the United States of 22,000 cases. However, CSD occurs in persons of all ages, the highest age-
specific incidence is among children aged <10 years. Infection with *B. henselae* is one of the most common causes of chronic lymphadenopathy among children, and in some case series up to 25% of the these infections result in severe systemic illness. Because TCH is a referral hospital, the frequency of severe manifestations seen in this series is probably disproportionately high relative to general practice. Other serious manifestations of CSD not included in this series are granulomatous conjunctivitis, neuroretinitis, and atypical pneumonia. In immunocompromised persons, *B. henselae* infections can cause other potentially life-threatening disease manifestations (e.g., bacillary angiomatosis and peliosis).

Serologic testing is the standard method of diagnosis and should be considered for patients who present with adenopathy, fever, malaise, and history of feline contact. A single elevated indirect immunofluorescence assay titer or enzyme immunoassay value for IgG or IgM antibodies are generally sufficient to confirm CSD, because initiation of a humoral immune response generally precedes or is concurrent with symptom onset (4). IgG levels rise during the first 2 months after onset of illness, followed by a gradual decline. Other diagnostic assays, including polymerase chain reaction and bacterial culture, are available on a more limited basis at reference laboratories.

Treatment recommendations for *Bartonella*-associated diseases, including CSD, depend on the specific disease presentation. For most forms of CSD, assessing the efficacy of various antibiotics is difficult because symptoms are generally self-limiting over time, even in the absence of specific therapy. Recent experience with azithromycin suggests that this antibiotic hastens resolution of adenopathy of CSD. For patients with more severe disease, other antibiotic regimens have been successful, including azithromycin or doxycycline in combination with rifampin or rifampin alone; doxycycline or erythromycin are considered the drugs of choice for bacillary angiomatosis and peliosis.

CSD predominantly occurs in fall and winter because of either seasonal fluctuations in zoonotic transmission between felines or temporal changes in animal behavior and reproduction. Cat fleas (*Ctenocephalides felis*) are involved in the transmission of *B. henselae* among cats, but the role of fleas or other arthropods in the transmission of this pathogen to humans is not known. Scratches, licks, and bites from domestic cats, particularly kittens, are important risk factors for infection. Recommendations for prevention of CSD include vigilant elimination of fleas from feline pets and avoidance of traumatic injury from cats for persons who are immunocompromised or who have heart-valve abnormalities. Cats rarely demonstrate overt signs of illness from infection, and no vaccines are commercially available to prevent *B. henselae* infection in animals.

**REFERENCES**

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**Trends in Deaths From Systemic Lupus Erythematosus—United States, 1979-1998**

*MMWR.* 2002;51:371-374

1 figure, 1 table omitted

**SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)** is a serious autoimmune disease of unknown etiology that can affect several organs. Because SLE affects connective tissues and because painful joints and arthritis are among its most common manifestations, this disease is classified with arthritis and other rheumatic diseases. SLE is one of the more fatal forms of rheumatic diseases and non-Caucasian race is a risk factor for death from SLE; however, trends in death from SLE have not been analyzed recently. To characterize deaths from SLE, CDC reviewed SLE deaths during 1979-1998. This report presents the results of that analysis, which indicate that marked age-, sex-, and race-specific disparities exist in SLE death rates and that death rates have increased by approximately 70% during the study period among black women aged 45-64 years. Prevention of deaths requires early recognition and diagnosis of SLE and appropriate therapeutic management.

The analysis used National Center for Health Statistics Multiple Cause-of-Death Public Use Data Tapes for 1979-1998. These national mortality statistics were based on data from death certificates filed in state vital statistics offices. Demographic data (e.g., age and race/ethnicity) listed on death certificates were reported by funeral directors, usually from information provided by the decedent’s family. Causes of death listed on death certificates were reported by a physician, medical examiner, or coroner by using a format specified by the World Health Organization and endorsed by CDC. An SLE death was defined as any death of a U.S. resident coded with an underlying cause of death of systemic lupus erythematosus (International Classification of Diseases, Ninth Revision, code 710.0). Death rates were calculated by using annual deaths and corresponding U.S. resident population estimates. Death rates were calculated for whites and blacks. Rates for other races were not calculated because numbers were too small for meaningful analysis.

During 1979-1998, the annual number of deaths increased from 879 to 1,406, and the crude death rate increased from 39 to 52 per 10 million population, with 22,861 deaths reported during the study period. Of all SLE deaths, 36.4% occurred among persons aged 15-44 years. For each year, crude death rates increased with age, were 3 times higher among women than men, and were >3 times higher among blacks than whites. Among black women, death rates were highest and increased most (69.7%) among those aged 45-64 years, with little difference in rates among other age groups.
A higher incidence of SLE among black women might account for the racial differences in death rates. However, no ongoing population-based studies exist that determine how changes in SLE incidence contributed to the increase in the death rate. Beyond a change in incidence, other remediable reasons for an increase in SLE mortality among black women include later diagnosis, problems in access to care, less effective treatments, and poorer compliance with therapeutic regimens.  

The findings in this report are subject to at least four limitations. First, death rates might be underestimated. Because multiple cause-of-death data were used in this analysis, other causes of death (e.g., kidney disease and heart disease) might have been listed as the underlying cause of death rather than SLE. An additional 17,450 persons who died during 1979-1998 had SLE listed as an associated cause of death on their death certificates. Second, SLE can be difficult to diagnose clinically, and both underdiagnosis and overdiagnosis (e.g., because of positive antinuclear antibody tests) occur.  

However, physicians reporting SLE as the underlying cause of death presumably had sufficient data supporting the diagnosis to cite SLE first instead of other causes. Third, rates for racial/ethnic populations other than white and black were not calculated because numbers were too small for meaningful analysis. These populations might have high rates of SLE. Finally, because prevalence estimates for SLE are variable, population death rates were calculated rather than case fatality rates.

Arthritis and other rheumatic conditions are highly prevalent, disabling, and costly. SLE accounts for 14.5% of all deaths from arthritis (CDC, unpublished data, 1997) and represents one of these conditions that has premature mortality; approximately one third of deaths from SLE occur among persons aged <45 years. Of all deaths from arthritis, SLE accounts for 44.0% of deaths among persons aged <45 years (CDC, unpublished data, 1997).

Because of SLE’s protean manifestations, preventing excess and premature deaths will require clinical suspicion of the diagnosis, early recognition, appropriate therapeutic management, compliance with treatment, and improved treatment of long-term consequences (e.g., renal disease or accelerated atherosclerosis). 

One of the public health strategies outlined in The National Arthritis Action Plan is to better define issues related to rheumatic conditions such as SLE. Because further research into the causes of the marked age-, sex-, and race-specific disparities in death rates and temporal changes in death rates is necessary, CDC plans to develop a large population-based registry of SLE to monitor trends in SLE incidence and prevalence and better characterize persons with this disease. Studies conducted from this registry will examine why disparities and death rates exist and how mortality from SLE can be reduced.

REFERENCES