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Outbreak of Powassan Encephalitis—Maine and Vermont, 1999-2001

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POWASSAN (POW) VIRUS, A NORTH American tickborne flavivirus related to the Eastern Hemisphere's tickborne encephalitis viruses,[1] was first isolated from a patient with encephalitis in 1958.[1,2] During 1958-1998, 27 human POW encephalitis cases were reported from Canada and the northeastern United States.[3] During September 1999-July 2001, four Maine and Vermont residents with encephalitis were found to be infected with POW virus. These persons were tested for other arbovirus infections found in the northeast after testing for West Nile virus (WNV) infection was negative. This report describes these four cases, summarizes the results of ecologic investigations, and discusses a potential association between ticks that infest medium-sized mammals and the risk for human exposure to POW virus. The findings underscore the need for personal protective measures to prevent tick bites and continued encephalitis surveillance.

Case Reports

Case 1. In June 2001, a 70-year-old man from Kennebec County, Maine, was taken to a local hospital with generalized muscle weakness, somnolence, diarrhea, and anorexia. On clinical examination, he had a fever of 104.7°F (40.4°C), leukocytosis of 11,500/mm^3 (normal: 4,300-10,800/mm^3), decreased renal function, and anemia. He subsequently developed left-sided hemiplegia and marked confusion. Cerebrospinal fluid (CSF) contained 40 white blood cells (WBCs)/mm^3 (normal: $<$4/mm^3) (87% lymphocytes) with elevated protein (96 mg/dL; normal: 20-50 mg/dL). Magnetic resonance imaging (MRI) revealed parietal changes consistent with microvascular ischemia or demyelinating disease. No causes for his apparent stroke were found. After 22 days of hospitalization, he was discharged to a rehabilitation facility. Nearly 3 months after symptom onset, he remains in the facility and is unable to move his left arm or leg. Serum specimens and CSF collected 3 days after hospitalization revealed POW virus-specific IgM; neutralizing antibody (1:640 titer) also was found in serum specimens. Although some cross-reaction with WNV and St. Louis encephalitis (SLE) virus occurred in the IgM assay, no neutralizing antibody was found.

The patient had not left Maine for 25 years. On ecologic investigation, overgrown bushes, leaf piles, and stacks of old lumber and scrap metal covered his property. Family members reported seeing woodchucks, skunks, and squirrels on the property. During the 2 weeks before illness, the patient's main activities were lying on the ground repairing a boat hull and yard work. Approximately 6 weeks after illness onset, nine medium-sized mammals were trapped on or near the patient's property. Collections from these mammals and the grassy and brushy areas of the property yielded 31 ticks (*Ixodes cookei*). Tests for POW virus infection were conducted at CDC. Of the nine mammal serum samples, four (two woodchucks and two skunks) contained neutralizing antibody to POW virus, but no virus was isolated from the ticks.

Case 2. In September 2000, a 53-year-old woman from York County, Maine, sought medical care at a local hospital for loss of balance, visual disturbance, and fever of 103°F (39.4°C). Her clinical examination showed agitation without confusion, ataxia, bilateral lateral gaze palsy, and dysarthria. CSF contained 148 WBCs/mm^3 (46% neutrophils, 40% lymphocytes). During hospitalization, she developed altered mental status, generalized muscle weakness, and complete ophthalmoplegia. An electroencephalogram (EEG) indicated diffuse encephalitis, and a MRI showed bilateral temporal lobe abnormalities consistent with microvascular ischemia or demyelinating disease. After 13 days, she was transferred to a rehabilitation facility where she remained for 2 months. Nine months after onset of symptoms, she was walking and had regained her strength, but the ophthalmoplegia continued. A serum specimen collected 19 days after illness onset was positive for POW virus-specific IgM and neutralizing antibody (1:640 titer) and negative for WNV and SLE virus antibodies.

The patient had not left Maine in several months before illness onset. During two visits to a rural vacation home in the month before illness onset, the patient removed several squirrel nests but reported no contact with ticks or rodents. One month after illness onset, an ecologic evaluation of her primary home noted a well-manicured suburban property near brush and woodlands. No evidence of medium-sized mammals was found, and only three *Ix. scapularis* were collected; no POW virus was isolated. Nine months after illness onset, an ecologic evaluation of the patient's vacation home found several mammals, but none had ticks, and no serology samples were collected.

Case 3. In July 2000, a 25-year-old man from Waldo County, Maine, sought medical care at a local hospital for fever of 101.3°F (38.5°C), headache, vomiting, somnolence, and confusion. On clinical examination, the patient had difficulty answering simple questions and was intermittently uncooperative. He had bilateral hand twitching, muscle weakness, and pronounced lip smacking. CSF contained 920 WBCs/mm^3 (74% lymphocytes) with elevated protein (77 mg/dL). EEG showed diffuse background slowing consistent with encephalitis. After 11 days of hospitalization, he was transferred to a rehabilitation facility. When discharged home 44 days later, the patient required assistance to stand and perform daily activities. Serum specimens and CSF collected 3 days after illness onset were negative for antibody to WNV and SLE virus but positive for POW virus-specific IgM antibody. The serum sample also had neutralizing antibody (1:80 titer) to POW virus. At the time of illness onset, the patient worked as a logger and lived in rural Maine where he raised livestock.

Case 4. In September 1999, a 66-year-old man from Washington County, Vermont, sought medical care at a hospital for somnolence, severe headache, increasing confusion, and bilateral leg weakness that developed over 6 days. On clinical examination, he was afebrile but had slow speech, memory loss, a wide-based gait, and bilateral weakness in proximal lower extremities. CSF contained 54 WBCs/mm^3 (95% lymphocytes) and elevated protein (67 mg/dL). An EEG showed diffuse background slowing consistent with encephalitis. When discharged home 11 days later, he could walk but had cognitive difficulties, including severe memory lapses. Serum specimens collected 19 days after illness onset contained POW virus-specific IgM and neutralizing antibody (1:640 titer) but no antibody to WNV and SLE virus. During the month before illness onset, the patient traveled frequently to a vacation home where he saw numerous squirrels and skunks.

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CDC Editorial Note: These four cases of POW encephalitis are the first reported in Maine and Vermont and the first in the United States since 1994.[4] Since the introduction of WNV into the northeastern United States in 1999,[5] testing for POW virus and other arboviruses that cause encephalitis has increased (CDC, unpublished data, 2001). These cases were identified as a direct result of requests for WNV testing. As surveillance continues, knowledge of the epidemiology of POW virus in the United States may increase.

In North America, POW virus has been isolated from four tick species, including *Ix. cookei, Ix. marxi, Ix. spinipalpus*, and *Dermacentor andersoni*; a variant POW virus also has been isolated from *Ix. scapularis;* and evidence of infection has been found in 38 mammal species, primarily woodchucks.[1,6] Unlike *Ix. scapularis*, the primary vector for Lyme disease, *Ix. cookei* rarely search for hosts on vegetation and are often found in or near the nests or burrows of medium-sized mammals. Infections have occurred from May to December, with a peak during June-September when ticks are most active.[1] Although neither the first or second patients recalled tick bites, ecologic investigations suggest that their illnesses resulted from visiting or living in areas where ticks are common. As with many infectious agents transmitted by *Ixodid* ticks, few infected persons recalled tick bites because these ticks are small and can be easily missed.[3]

POW encephalitis is associated with significant long-term morbidity and has a case-fatality rate of 10%-15%.[1,3] Because there is no vaccine or specific therapy for POW encephalitis, the best means of prevention is protection from tick bite. This includes using insect repellents, wearing light-colored clothing with long sleeves and pants tucked into socks or boots, avoiding or clearing brushy areas, and removing ticks before they attach or as soon after attachment as possible. Checking family pets also can prevent ticks from entering the home. Because *Ix. cookei* are often found on woodchucks and skunks and may be the primary vector of POW virus, environmental controls reducing human contact with small and medium-sized mammals should reduce risk for exposure to POW virus-infected ticks. Persons should keep areas adjacent to their home clear of brush, weeds, trash, and other elements that could support small and medium-sized mammals. When removing rodent nests, avoid direct contact with nesting materials and use sealed plastic bags for disposal and to prevent direct contact with ticks.

Because of the lack of awareness and the need for specialized laboratory tests to confirm diagnosis, the frequency of POW encephalitis may be greater than previously suspected. POW encephalitis should be included in the differential diagnosis of all encephalitis cases occurring in the northern United States, especially the northeast. Laboratory tests for POW virus infection are not commercially available but can be requested through state public health laboratories for testing at CDC. Awareness should be promoted among clinicians and public health staff, and tick-bite prevention strategies emphasized for the general public.

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Shigellosis Outbreak Associated With an Unchlorinated Fill-and-Drain Wading Pool—Iowa, 2001

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ON JUNE 15, 2001, LOCAL PHYSICIANS REported 11 cases of diarrhea to a county health department. Stool samples from two of these persons were culture confirmed as *Shigella sonnei*; one person was hospitalized. A preliminary investigation found that nine of these persons recently had visited a large city park with a wading pool. The Iowa Department of Public Health was asked to assist in an investigation of this outbreak. This report summarizes the results of the investigation, which implicated the inadequately disinfected wading pool as the source of the outbreak and presents strategies for preventing such outbreaks.

Beginning on June 15, telephone interviews were conducted using a questionnaire that included information about demographics, illness history, participation in group gatherings, water activities, and use of the park or wading pool. Ill persons were asked to identify others who were at the park or had similar symptoms. A primary case was defined as self-reported diarrhea in a person within 72 hours of visiting the park during June 11-13. A secondary case was defined as self-reported diarrhea in a person within 72 hours of household contact with a primary case-patient.

Of 89 persons interviewed, 69 met one of the case definitions. Of these, 45 (65%) were categorized as primary cases and 24 (35%) as secondary cases. Stool samples from 16 primary case-patients and 10 secondary case-patients were laboratory confirmed as *S. sonnei*, and all 26 isolates were indistinguishable by pulse field gel electrophoresis (PFGE). Of 24 isolates tested at a clinical laboratory, 16 (67%) were resistant to ampicillin and sensitive to trimethoprim-sulfamethoxazole, cefotaxime, and levofloxacin.

Illness onset among primary case-patients occurred during June 12-14. The median age was 6 years (range: 1-31 years); 23 (51%) were female. Symptoms included diarrhea (100%), nausea (51%), vomiting (47%), bloody diarrhea (39%), and headache (29%). Seven (16%) patients were hospitalized. Pool exposure was associated significantly with illness (risk ratio = 5.7; 95% confidence interval = 1.6-20.4). Illness onset among the 24 secondary case-patients occurred during June 15-22. The median age was 24 years (range: 0-63 years); 14 (58%) were female.

The pool, which has been in operation for approximately 60 years, is 40 feet in diameter, has a maximum depth of 14 inches, and has a 9400-gallon capacity. It is frequented by diaper- and toddler-aged children and as many as 20-30 children may be in the pool at one time. The pool is a "fill-and-drain" system and is filled each morning with potable city water through a direct inlet pipe and a centrally located fountain; it is drained and left empty each evening. The pool includes a backflow device but has no recirculation or disinfection system (i.e., pump, filter, or mechanical disinfection system). Each morning before filling, the pool is rinsed with a high-pressure washer and is scrubbed with a chlorine cleanser twice weekly. However, chlorine levels were not monitored and chlorine was not added to the pool water. Samples from the pool and other water sources in the park, including drinking fountains and faucets, were collected on June 15 and tested by the Colilert test, a rapid procedure to determine the presence of fecal coliforms. One pool sample tested positive for fecal coliforms and *Escherichia coli*. The pool was closed on June 15.

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CDC Editorial Note: In this outbreak, the drain-and-fill pool contained municipal water (0.4-0.5 ppm free available chlorine) with no subsequent chlorination so that the pool was probably unchlorinated for most of the time it was in use. Inadequate disinfection of this pool, combined with heavy use by diaper- and toddler-aged children, who are often incontinent and may have an increased prevalence of enteric infections, created a favorable environment for transmission of shigellosis.

Transmission of shigellosis over several days may have been a result of the residual contaminated water left in the pipes after draining the pool and persons with diarrhea visiting the pool on subsequent days. The infectious dose for *Shigella*[1] is low; as a result, a small volume of ingested water can cause infection. The lack of chlorination that led to transmission of shigellosis in this wading pool also increased the risk for spreading life-threatening pathogens such as *E. coli* O157:H7.

This outbreak together with surveillance data that suggest an increase in disease outbreaks associated with recreational water exposure[2] illustrate the need for strict adherence of recreational water venues to existing health codes, enforcement of these codes, and education of pool operators about adequate disinfection and maintenance of pool water quality. Improved facility design and adequate water treatment can decrease the risk for transmission of illness. In addition to improved pool design and improved management and maintenance, increased education of pool staff and the public about the potential for spreading recreational water illness and development of strategies for reducing the spread of swimming-related illness is crucial to decreasing transmission.[3]

Swimming is a shared water activity that can result in disease transmission, even with adequate chlorination, when water becomes contaminated and is subsequently swallowed. Strategies for prevention include (1) not swimming when ill with diarrhea, (2) not swallowing recreational water, and (3) practicing good hygiene when using a pool. Parents should take children on bathroom breaks regularly, use appropriate diaper changing areas, wash hands after using the toilet or changing diapers, and shower before entering the pool. Swim pants and diapers do not prevent leakage of diarrhea; therefore, they are not an acceptable solution for a child with diarrhea and are not a substitute for frequent diaper changing.

Approximately 10,000 cases of *S. sonnei* are diagnosed each year in the United States, and most occur in young children.[4] Subsequent to the outbreak described in this report, a community-wide outbreak of shigellosis involving several local day care centers occurred; PFGE patterns were identical for both swimming-related and community-outbreak isolates. The ease with which single outbreaks can expand into communitywide outbreaks of *S. sonnei*[5] underscores the importance of educating the community about potential modes of transmission (e.g., child care facilities, food handlers, and swimming) and the implementation of appropriate prevention recommendations during outbreaks (e.g., thorough hand washing after using restrooms, changing diapers, and before handling/preparing food, enforcement of exclusion criteria at child care facilities, and exclusion of persons from swimming while ill with diarrhea). Child care facilities should follow strict hygiene recommendations, including supervised hand washing for young children, and may consider refraining from using water play tables and inflatable pools that may lead to transmission. In addition, communication with pool operators about ongoing outbreaks may improve vigilance in maintaining disinfectant levels necessary to reduce the risk for transmission among bathers at community pools. Additional information about preventing recreational water illness is available at http://www.healthyswimming.org.[3]

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Outbreaks of Multidrug-Resistant *Salmonella* Typhimurium Associated With Veterinary Facilities—Idaho, Minnesota, and Washington, 1999

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CDC RECEIVED REPORTS IN 1999 FROM three state health departments of outbreaks of multidrug-resistant *Salmonella* serotype Typhimurium infections in employees and clients of small animal veterinary clinics and an animal shelter. Salmonella infections usually are acquired by eating contaminated food; however, direct contact with infected animals, including dogs and cats, also can result in exposure and infection.[1] This report summarizes clinical and epidemiologic data about these outbreaks and reviews methods of reducing the likelihood of Salmonella transmission in veterinary settings by avoiding fecal-oral contact.

Idaho

During September-October, the Idaho Department of Health and Welfare identified through routine surveillance an outbreak of *Salmonella* infections among employees of a small animal veterinary clinic; 10 of 20 persons had abdominal cramps and diarrhea, and two of the 10 had bloody diarrhea. The median age of the ill persons was 31 years (range: 19-44 years), the median duration of illness was 7 days (range: 4-12 days), and four persons sought medical care. The index patient reported caring for several kittens with diarrhea 1 or 2 days before illness onset; stool specimens were not cultured and the kittens died. All 10 ill employees ate meals in the clinic and had no common exposures outside the clinic. Stool specimens from five ill employees yielded *S.* Typhimurium. All isolates were indistinguishable by pulsed-field gel electrophoresis (PFGE); reacted to phage but did not conform to a definitive phage type; and were resistant to ampicillin, ceftriaxone, cephalothin, chloramphenicol, clavulanic acid/amoxicillin, gentamicin, kanamycin, streptomycin, sulfamethoxazole, and tetracycline.

Minnesota

The Minnesota Department of Health (MDH) routinely receives animal *S.* Typhimurium isolates from the Minnesota Veterinary Diagnostic Laboratory. In 1999, MDH tested *S.* Typhimurium isolates from nine cats and seven humans that were indistinguishable by PFGE. All isolates were resistant to ampicillin, chloramphenicol, streptomycin, sulfamethoxazole, and tetracycline (R-type ACSSuT). Three cat and two human isolates tested were definitive type (DT) 104. The cats had died in an animal shelter during September-October at age 6-14 weeks as a result of infection. The median age of ill persons was 6 years (range: 11 months-23 years), and the median duration of diarrhea was 8 days (range: 5-11 days); all persons sought medical care, and one was hospitalized for 4 days. An adult treated with ciprofloxacin shed *S.* Typhimurium in stool at least 214 days after illness onset.

A connection with the animal shelter was established for six of the seven human patients; four purchased cats from the shelter during August-

October and two attended the same day-care center as an ill child who owned a cat from the shelter. One cat developed bloody diarrhea 1 day after adoption and onset of illness in the patient began 4 days later. Two cats remained asymptomatic; however, the owner became ill 77 days after adopting the cats. The outbreak strain of *S.* Typhimurium was recovered from one cat 115 days after adoption.

Washington

Through laboratory-based surveillance and patient interviews, the Washington State Department of Health detected in late 1999 an outbreak of *Salmonella* infections associated with a small animal veterinary clinic. Stool specimens from three ill persons yielded *S.* Typhimurium, all three sought medical care, but none was hospitalized. One ill person was a clinic employee and the two others recently had brought their cats to the clinic, one for elective surgery and the other for a urinary tract infection. The cats developed diarrhea after their discharge from the clinic and the owners subsequently became ill. The clinic was the only common exposure reported by the three ill persons. *S.* Typhimurium was isolated from 14 cats associated with this clinic; some of the cats initially presented with diarrhea. Isolates from ill persons and cats were indistinguishable by PFGE. All isolates were DT104 R-type ACSSuT.

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CDC Editorial Note: Although most of the estimated 1.4 million *Salmonella* infections that occur each year in the United States are transmitted through food, *Salmonella* also is transmitted through exposure to contaminated water, reptiles, farm animals, and pets.[1] It is unknown how the human patients in these outbreaks became infected with *Salmonella*; however, the inadvertent ingestion of animal feces or food contaminated with animal feces may have occurred as the result of suboptimal sanitation and hygienic practices in the veterinary facilities. Many cats in these facilities had a diarrheal illness that also may have contributed to *Salmonella* transmission. Even after recovery from an acute episode of *Salmonella* gastroenteritis, fecal shedding of *Salmonella* can occur and may last several months. In addition, the use of antimicrobial agents in veterinary facilities may have contributed to transmission of multidrug-resistant *Salmonella* by lowering the infectious dose needed for ingestion to cause illness in animals and increasing the likelihood of transmission to humans. Although outbreaks of multidrug-resistant *Salmonella* with human and animal illness have been reported in large animal veterinary facilities (e.g., horse clinics),[2,3] outbreaks associated with small animal facilities are rare. The outbreaks described in this report demonstrate that small animals shed *Salmonella* and that small animal facilities can serve as foci of transmission for *Salmonella* to other animals and humans.

In 1999, the most commonly isolated *Salmonella* serotype in the United States was *S.* Typhimurium, accounting for 23% of laboratory-confirmed *Salmonella* cases.[4] Multidrug resistance among *S.* Typhimurium isolates is common; of human *S.* Typhimurium isolates received at CDC through the National Antimicrobial Resistance Monitoring System, 46% were multidrug-resistant; 61% of these were R-type ACSSuT and 23% were R-type AKSSuT (resistant to ampicillin, kanamycin, streptomycin, sulfamethoxazole, and tetracycline). R-type ACSSuT and R-type AKSSuT, the two most common multidrug-resistant Salmonella strains, accounted for 7% and 3% of non-Typhi *Salmonella* isolates, respectively.[5] Investigations in the United States have found associations between human infections caused by R-type ACSSuT and R-type AKSSuT strains of *S.* Typhimurium and contact with cattle, including eating and drinking unpasteurized dairy products.[6,7] *S.* Typhimurium DT104 R-type ACSSuT has been associated with contact with pets in the United Kingdom[8]; outbreaks described in this report are the first to associate DT104 with pets in the United States.

To prevent salmonellosis, persons should wash their hands before eating and after handling food. Immunosuppressed persons should avoid animals aged <6 months and animals with diarrhea.[9] Veterinary workers should wash their hands after handling pets, especially after handling feces. These workers can further reduce their exposure to feces by wearing rubber or disposable gloves, and by removing gloves and washing their hands immediately after finishing a task that involves contact with animal feces. Although there have been no reports of *Salmonella* transmission through splash exposures, workers might consider taking measures to reduce splashes of feces to the mouth when hosing or cleaning a kennel. All surfaces contaminated with feces should be cleaned and disinfected. No eating should be allowed in animal treatment or holding areas. Because use of antimicrobial agents contributes to increasing resistance and facilitates transmission of multidrug-resistant *Salmonella*, eliminating inappropriate use of antimicrobial agents may help to prevent outbreaks of multidrug-resistant *Salmonella* infections in veterinary facilities.[10]

REFERENCES*

10 available

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