Epidemiology of Measles—United States, 1998

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DURING 1998, A PROVISIONAL TOTAL OF 100 confirmed measles cases was reported to CDC by state and local health departments, representing a record low number of cases and 28% fewer than the 138 cases reported in 1997.1 This report describes the epidemiology of measles during 1998, which suggests that measles is no longer an indigenous disease in the United States.

Case Classification
Measles cases among persons who were infected outside the United States are classified as internationally imported cases. Cases among persons who were infected in the United States are classified as indigenous measles cases. Indigenous cases are subclassified into three groups: cases epidemiologically (epi)-linked to importation (a chain of transmission caused by an internationally imported case); imported virus cases (a chain of transmission from which an imported measles virus strain was isolated but a link to an internationally imported case was not identified); and not importation-associated cases (no epidemiologic or virologic association to importation was detected). Internationally imported cases, cases epi-linked to importation, and imported virus cases are all considered importation-associated cases.

Of the 100 cases reported, 26 were internationally imported, and 74 were indigenous. Of the 74 indigenous cases, 45 were importation-associated, and 29 were not importation-associated. The proportion of cases not associated with importation has declined from 85% in 1995, 72% in 1996, 41% in 1997, to 29% in 1998. The 45 importation-associated indigenous cases included 13 epi-linked cases and 32 imported virus cases.

All 32 imported virus cases occurred in an outbreak in Alaska, which started 4 weeks after an imported case of measles was diagnosed in a visitor from Japan. Measles virus isolated from cases in this outbreak was nearly identical to virus circulating in Japan, although no virus was cultured from the imported case and no epidemiologic link between the imported case and the outbreak was detected.3 In addition to the strain isolated from the Alaska outbreak, viral genomic sequencing of specimens from epi-linked cases allowed genotype classification of measles virus strains from six chains of transmission epidemiologically linked to internationally imported cases. Virus strains isolated from cases in New York, Vermont, California, Massachusetts, and Washington matched viral genotypes from Germany, Cyprus, Japan, China, and Croatia, respectively. Measles virus was isolated from the Indiana outbreak but genotype information was unavailable from Zimbabwe, the source country of the imported case.

Internationally Imported Measles Cases
The 26 internationally imported cases reported in 1998 represent the lowest number of imported cases since the recording of importation status began in 1983. Imported cases from the Americas remained at very low levels, and imported cases from Europe and Asia declined compared with the previous 4 years. India, Japan, Kenya, Pakistan, and Saudi Arabia each were the source of two imported cases. One importation was reported from each of the other countries. Of 26 imported cases, 14 occurred among international visitors and 12 occurred among U.S. residents exposed to measles while traveling abroad.

Geographic Distribution
During 1998, 28 states and the District of Columbia reported no confirmed measles cases, compared with 21 states in 1997. Eight states accounted for 82% of cases: Alaska (33 cases), Arizona (11), Michigan (10), California (nine), New Jersey (eight), New York (four), Pennsylvania (four), and Indiana (three). In the remaining 14 states, two or fewer cases were reported. Eight states reported indigenous measles cases not associated with importation.

Temporal Patterns of Transmission
The median number of cases per week was one (range: 0-11). During 35 weeks, all reported measles cases were importation-associated, including 21 consecutive weeks (weeks 25-45). Half of the indigenous cases that were not importation-associated occurred in two outbreaks: in New Jersey (weeks 13-16) and in Michigan (weeks 20-23).

Age and Vaccination Status
The age distribution and vaccination status of U.S. residents with measles differed from those of international visitors. Most U.S. residents with measles had been vaccinated with one or more doses of measles vaccine (53%), and 86% of international visitors with measles were unvaccinated.

Outbreaks
Six measles outbreaks were reported in 1998, the fewest ever reported to CDC. Outbreaks occurred in Alaska (33 cases), Arizona (11), Michigan (nine), New Jersey (six), Indiana (three), and Pennsylvania (three). The 65 measles cases reported from these outbreaks represented 65% of all cases reported during 1998. The ages of persons with outbreak-associated cases ranged from 5 months to 44 years (median: 15 years).

The largest measles outbreak reported since 1996 occurred in a high school in Anchorage, Alaska; 30 of the
33 cases had received one dose of measles vaccine. A 4-year-old unvaccinated Japanese child visiting Anchorage had measles diagnosed 4 weeks before the other cases in the outbreak. No epi-link was reported between this case and subsequent cases. However, the genotype of viral RNA collected from outbreak cases was nearly identical to virus circulating in Japan. The interval from the onset of rash in the imported case to the end of the outbreak was 15 weeks (August 10 to November 19, the longest interval of transmission in 1998). As a result of the outbreak, the Alaskan Health Department now requires two doses of measles vaccine for all students in grades K-12. Three outbreaks (Arizona, Indiana, and Pennsylvania) were epi-linked to an imported measles case, and two outbreaks (Michigan and New Jersey) were not importation-associated.

CDC Editorial Note: Analysis of epidemiologic data for 1998 suggests measles is no longer an indigenous disease in the United States. Most cases reported in 1998 were associated with importation, including the short chains of indigenous transmission of measles that occurred following international importation of measles.

Cases not associated with importation were insufficient to represent a continuous indigenous chain of measles transmission and probably were misclassifications (not measles), associated with undetected imported measles cases, or linked to known imported cases through chains of transmission not detected by the surveillance system. Misclassifications resulting from false-positive laboratory tests are an expected result of intensive investigation for a rare disease using a laboratory test that is not 100% specific.

Some cases may spread from undetected imported cases of measles. Detecting imported cases is difficult. International visitors with measles may leave the country before the rash appears or before they seek medical care. Even when the imported case is detected, it is difficult to detect every case in the chain of transmission, as was seen in the outbreak in Alaska. This highlights the need to obtain viral specimens from every chain of transmission to supplement epidemiologic information.

The largest outbreak in 1998 occurred in a high school without a second dose measles vaccine requirement. As of the 1998-99 school year, 55% of U.S. students were required by their states to have two doses of measles vaccine (CDC, unpublished data, 1998). Vaccination of all students with two doses of measles vaccine by 2001, as recommended by the American Academy of Pediatrics and CDC’s Advisory Committee on Immunization Practices, will reduce future school outbreaks. Completion of this strategy should further decrease the risk for indigenous transmission of measles following importation of the measles virus.

The United States appears to have eliminated measles as an indigenous disease. High measles vaccination coverage and strong surveillance remain critical to preventing international imported measles cases from causing a resurgence of measles in the United States.

REFERENCES

5 available

Three or more cases in a single chain of transmission.

Meningococcal Disease—New England, 1993-1998

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1 figure, 1 table omitted

NEISSERIA MENINGITIDIS, A LEADING CAUSE of bacterial meningitis and sepsis in children and young adults in the United States, causes both sporadic disease and outbreaks. Preventing and controlling meningococcal disease remains a public health challenge because of the multiple serogroups and the limitations of available vaccines. Vaccination with the polysaccharide meningococcal vaccine, which protects against serogroups A, C, Y, and W135 of N. meningitidis, is recommended by the Advisory Committee on Immunization Practices (ACIP) for controlling outbreaks but routine vaccination is not recommended for control of sporadic cases. During 1998, a cluster of meningococcal disease cases occurred in Rhode Island, and although the situation did not meet ACIP criteria for an outbreak, the Rhode Island Department of Health recommended vaccination of all residents aged 2-22 years. This action stimulated controversy in Rhode Island and the rest of New England (Connecticut, Maine, Massachusetts, New Hampshire, and Vermont) and prompted a review of the epidemiology of meningococcal disease in the region. This report describes meningococcal disease data reported to the region’s state health departments during 1993-1998 and discusses the situation in Rhode Island.

Surveillance. Connecticut and Massachusetts conducted prospective enhanced surveillance for meningococcal disease beginning in 1995 and 1996, respectively. In Rhode Island, additional case ascertainment was done in 1998 by reviewing hospital inpatient discharge data and hospital records for all confirmed and probable cases from 1992 through 1998. Surveillance in Maine, New Hampshire, and Vermont consisted of routine reporting for meningococcal disease. To calculate incidence, census data for 1996 were used.

Case Definition and Detection Method. A confirmed case of meningococcal disease was defined as isolation of N. meningitidis from a normally sterile site (e.g., blood or cerebrospinal fluid [CSF]) from a person with clinically compatible illness. A probable case of meningococcal disease was defined as purpura fulminans or detection of meningococcal polysaccharide antigen in CSF in the absence of a diagnostic culture from a person with clinically compatible illness.
Case Characteristics. During 1993-1998 in New England, 937 cases of meningococcal disease were reported. Of these, 899 (96%) met the definition for confirmed or probable meningococcal disease; 863 (96%) were confirmed by culture and 36 (4%) were probable cases. The proportion of confirmed cases varied by state from 100% (Vermont) to 84% (Rhode Island). Of the probable cases, 22 (61%) were reported as detection of meningococcal antigen in CSF, and 14 (39%) as purpura fulminans; 12 of 14 purpura fulminans cases were reported from Rhode Island. Of the 899 cases, 888 (99%) were considered primary (i.e., occurred in the absence of known close contact with another case-patient).1 The median age of all case-patients was 17 years (range: 3 days-98 years); 455 (51%) were female, and 88 case-patients died (case fatality rate [CFR] = 10%). The distributions of cases by age, sex, and serogroup were similar by state. Rhode Island had a significantly higher CFR (21%) (p = 0.001) than the other five states. Ten (<1%) cases were associated with outbreaks; the remainder was classified as sporadic disease.

Serogroups. Of the 758 (89%) cases with serogroup reported, 308 (41%) were serogroup C, 217 (29%) were serogroup Y, and 200 (26%) were serogroup B. Among case-patients with known serogroups, the proportion with serogroup Y meningococcal disease increased from 15% in 1993 to 43% in 1998 (p<0.005).

Incidence. During 1993-1998, the average annual reported incidence of meningococcal disease was 1.1 cases per 100,000 population. Annual incidence increased significantly from 0.9 cases per 100,000 population in 1993 to 1.4 cases per 100,000 population in 1997 (chi square for linear trend, p<0.001) and declined from 1.4 to 0.9 cases per 100,000 population from 1997 to 1998 (p<0.001). Excluding any state that did not alter this trend. The lowest disease rate reported was 0.4 cases per 100,000 population (New Hampshire and Rhode Island in 1993) and the highest rate was 2.5 cases per 100,000 population (Rhode Island in 1997). Age groups with the highest incidence were children ≤2 years (6.4 cases per 100,000) and young adults aged 15-19 years (3.0 cases per 100,000).

Reported by: RS Nelson, DVM, CA Morin, MPH, ML Carter, MD, P Mshar, JL Hadler, MD, State Epidemiologist, Connecticut Dept of Public Health. G Beckert, MPH, K Gensheimer, MD, State Epidemiologist, Maine Dept of Human Services. J Isadore, MPH, P Kludt, MPH, BT Matyas, MD, A DeMaria, Jr, MD, State Epidemiologist, Massachusetts Dept of Public Health. N Johnson, J Greenblatt, MD, State Epidemiologist, New Hampshire Dept of Health and Human Services. L Mouradian, P Nolan, MD, U Bandy, MD, State Epidemiologist, Rhode Island Dept of Health. JK Carney, MD, CJ Greene, MPH, PD Galbraith, DMD, State Epidemiologist, Vermont Dept of Health. Meningitis and Special Pathogens Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; Div of Applied Public Health Training, Epidemiology Program Office; and an EIS Officer, CDC.

CDC Editorial Note: Data in this report indicate that rates of meningococcal disease in New England increased during 1993-1997, then declined in 1998. The average annual rate in Rhode Island during this period was similar to rates in neighboring states. The rates also were similar to those reported in the United States during the same period.3,4 These changes in incidence probably represent natural fluctuations in disease incidence, changes in circulating strains of N. meningitidis, the population’s susceptibility to disease, or some combination of these variables.

Surveillance data indicated that the CFR among case-patients from Rhode Island were significantly higher than the CFR among case-patients from other states in the region. Twelve of 14 cases of purpura fulminans were reported from Rhode Island, and these case-patients had a higher CFR. However, when patients with purpura fulminans were eliminated from the analysis, the CFR in Rhode Island still remained elevated (20% versus 10%; p<0.003). Possible explanations for this difference in CFR include timing of antibiotic use and strain virulence. Some studies have reported that early antibiotic intervention is associated with reduced mortality; other studies have suggested that the finding may be attributable to confounding by variables such as host factors and severity of illness on presentation.5,6 In Rhode Island, case investigations have found that antibiotics were appropriately given, suggesting that other factors contributed to the high CFR.

Between November 26, 1997, and February 23, 1998, Rhode Island reported nine confirmed cases (four serogroup C, three serogroup Y, and two serogroup B) and three probable cases of meningococcal disease, with three deaths. Although this cluster did not constitute an outbreak as defined by ACIP guidelines,1 a statewide vaccination program for residents aged 2-22 years was initiated. Approximately 60%-70% of the targeted population received the vaccine. The precedent of an earlier vaccination campaign in Woonsocket in 1996 and an increased reported incidence in disease and CFR generated public and medical concern and social and political pressure that influenced the decision to vaccinate (P.A. Nolan, MD, Rhode Island Department of Health, personal communication, 1998). Information on meningococcal disease in Rhode Island is available on the World-Wide Web at http://www.health.state.ri.us/nemingo.htm.

Although some cases may be prevented by this approach, its overall impact may be limited for several reasons: it will not protect children aged <2 years, in whom rates of disease are highest; it does not protect against serogroup B disease, which accounts for 26% of disease in the region; and, because the vaccine does not affect carriage, it will not affect disease among the 30%-40% of the target population who chose not to be vaccinated. Monitoring of disease in Rhode Island over the next few years will allow further evaluation of this strategy.

During 1993-1998, <1% of cases in New England were classified as outbreak associated. Most cases of meningococcal disease were sporadic and therefore not preventable with strategies that target outbreaks. For efficacious protection of meningococcal disease in infants and young children, conjugate serogroup A, C, Y, and W135 meningococcal vaccines have been developed through methods similar to those used for Haemophilus influenzae.

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Carbon Monoxide Poisoning Deaths Associated With Camping—Georgia, March 1999

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Carbon monoxide (CO) is an odorless, colorless, nonirritating gas produced by the incomplete combustion of carbon-based fuels. CO exposure is responsible for more fatal unintentional poisonings in the United States than any other agent, with the highest incidence occurring during the cold-weather months. Although most of these deaths occur in residences or motor vehicles, two incidents among campers in Georgia illustrate the danger of CO in outdoor settings. This report describes the two incidents, which resulted in six deaths, and provides recommendations for avoiding CO poisoning in outdoor settings.

Cases 1-4. On the afternoon of March 14, 1999, a 51-year-old man, his 10-year-old son, a 9-year-old boy, and a 7-year-old girl were found dead inside a zipped-up, 10-foot by 14-foot, two-room tent at their campsite in southeast Georgia (a pet dog also died). A propane gas stove, still burning, was found inside the tent; the stove apparently had been brought inside to provide warmth. The occupants had died during the night. Postmortem carboxyhemoglobin (COHb) levels measured 50%, 63%, 69%, and 63%, respectively, in the four decedents (in the general U.S. population, COHb concentrations average 1% in nonsmokers and 4% in smokers [3]).

Cases 5 and 6. On March 27, 1999, a 34-year-old man and his 7-year-old son were found dead inside their zipped-up tent at a group camping site in central Georgia. They were discovered by other campers just before 9 a.m. A charcoal grill was found inside the tent; the grill apparently had been brought inside to provide warmth after it had been used outside for cooking. Postmortem COHb levels in the two campers measured 68% and 76%, respectively.

CDC Editorial Note: On respiration, CO binds to hemoglobin with an affinity 200-250 times greater than that of oxygen, forming a COHb complex.4 The principal toxic effect of CO exposure is tissue hypoxia because COHb is less efficient at transporting and delivering oxygen. Poisoning symptoms, such as headache, dizziness, and nausea, usually are seen at COHb levels of ≥10% in otherwise healthy persons.2

During 1979-1988 in the United States, from 878 to 1513 deaths per year were attributed to unintentional CO poisoning.1 CO poisoning has been reported in many different settings, including homes,3 automobiles,6 and indoor arenas.7 The findings in this report demonstrate the danger of CO from portable gas stoves and charcoal grills, specifically if placed inside a tent or other confined sleeping area. In the United States during 1990-1994, portable fuel-burning camp stoves and lanterns were involved in 10-17 CO poisoning deaths each year, and charcoal grills were involved in 15-27 deaths each year.2 During this same time, an annual average of 30 fatal CO poisonings occurred inside tents or campers.2

Evening temperatures often drop unexpectedly, even during warmer months of the year. Campers who are unprepared for colder weather may overlook the danger of operating fuel-burning camping heaters, portable gas stoves, or charcoal grills inside tents and campers. Camping stoves and heaters are not designed to be used indoors and can emit hazardous amounts of CO, and smoldering charcoal emits large amounts of CO. Inside a tent or camper, these sources produce dangerous concentrations of CO, which becomes even more dangerous to sleeping persons who are unable to recognize the early symptoms of CO poisoning.

To avoid hazardous CO exposures, fuel-burning equipment such as camping stoves, camping heaters, lanterns, and charcoal grills should never be used inside a tent, camper, or other enclosed shelter. Opening tent flaps, doors, or windows is insufficient to prevent build-up of CO concentrations from these devices. When using fuel-burning devices outdoors, the exhaust should not vent into enclosed shelters. Warnings about the potential for CO poisoning should be stated clearly in the owner’s manual and on labels permanently affixed to portable stoves. In 1997, changes made in the labeling requirements for retail charcoal containers more clearly conveyed the danger of burning charcoal inside homes, tents, or campers. Rather than relying on fuel-burning appliances to supply heat, campers should leave home with adequate bedding and clothing and should consume extra calories and fluids during the outing to prevent hypothermia. Continuing efforts to educate the public by organizations that promote outdoor activities or operate camping areas also should decrease camping-associated CO poisoning.

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