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Poliovirus Infections in Four Unvaccinated Children — Minnesota, August–October 2005

On September 29, 2005, the Minnesota Department of Health (MDH) identified poliovirus type 1 in an unvaccinated, immunocompromised infant girl aged 7 months (the index patient) in an Amish community whose members predominantly were unvaccinated for polio. The patient has no paralysis; the source of the patient's infection is unknown. Subsequently, poliovirus infections in three other children within the index patient's community have been documented. This report summarizes the ongoing investigation, provides information regarding poliovirus exposure risks and prevention measures in the United States, and offers recommendations to state health departments and clinicians.

Index Case Summary

The index patient was first admitted to a community hospital in central Minnesota for pneumonia in July 2005. Since August 22, this infant has been hospitalized continuously at three additional hospitals with failure to thrive, diarrhea, and recurrent infections. The infant was placed in strict isolation, and a diagnosis of severe combined immunodeficiency (SCID) was made on September 15. The infant is being clinically managed with intravenous immunoglobulin therapy and is being evaluated for bone marrow transplantation.

Laboratory Investigation

An enterovirus isolate from a stool specimen obtained on August 27, 2005, tested positive for a type 1 poliovirus at the MDH laboratory. Partial sequencing of the virus capsid protein coding region (VP1) of the poliovirus genome at the MDH laboratory identified it as a vaccine-derived poliovirus (VDPV). VDPVs are poliovirus strains derived from one of the three Sabin poliovirus strains in oral polio vaccine (OPV) that have $\geq 1\%$ difference in nucleotide sequence from the prototype vaccine virus (*I*). Additional sequencing of the entire poliovirus genome at the CDC polio laboratory confirmed that this strain was a VDPV, with 2.3% divergence in the VP1 region from the parent Sabin type 1 strain. The viral

genome demonstrates no recombination with other polioviruses or species C enteroviruses. Prospective serial stool samples from the infant are being tested to monitor ongoing infection and further mutations in the virus.

Epidemiologic Investigation

Because viral genomic data suggest this poliovirus might have been transmitted to the index patient from another immunocompromised person, the initial investigation focused on identifying immunodeficient persons among community contacts, health-care workers, and patients with whom the infant had potential contact before the first positive poliovirus culture on August 27. Staff and patient records at the hospitals are being reviewed, and inquiries are being made with community members and health-care providers.

Investigations also are under way at the four hospitals where the infant has been treated to determine whether nosocomial transmission from the infant has occurred. At the hospital where the infant currently is a patient, health-care workers and other staff members who have had exposure (without protection from contact precautions) to the infant or the infant's environment are being surveyed regarding polio vaccination status, immune status, and recent relevant illnesses in themselves and their family members. Stool samples are being obtained for viral cultures. Vaccination with inactivated polio vaccine (IPV) is being offered to health-care workers who might have been exposed or who have an ongoing risk for exposure and whose polio vaccination status is not up to date or is unknown. Stool specimens also are being obtained from potentially exposed patients at the hospital where the infant currently is a patient. At the first three hospitals where the infant was admitted, health-care workers are being surveyed regarding immune status and recent illness in themselves or their family members.

To examine community transmission of poliovirus, family members and others in the index patient's community are being surveyed regarding polio vaccination status, immune status,

and recent illnesses. To date, stool samples have been collected from 32 persons in five of 24 households, and serum samples have been obtained from eight persons in three households, including the index patient's household. Poliovirus type 1 has been confirmed in three of 32 stool specimens; partial sequencing of the VP1 region of these three isolates has indicated they also are VDPV type 1. The positive specimens were obtained from three unvaccinated siblings in one household (not the infant's household). None of these three children have been ill recently, and none were immunocompromised. Stool and serum samples are being requested from additional members of the community. Extended family members and community contacts from other areas who might have come into contact with the index patient are being identified and monitored for illness. IPV is being offered to community members who are not fully vaccinated for polio or whose polio vaccination status is unknown. Hospitals that serve this community and similar communities are being contacted, and retrospective and prospective surveillance is identifying patients whose diagnoses indicate conditions that are clinically consistent with poliovirus infection, including acute flaccid paralysis (AFP), Guillain-Barré Syndrome (GBS), transverse myelitis, and viral or aseptic meningitis.

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Editorial Note: The findings in this report are the first identification of a VDPV in the United States and the first occurrence of VDPV transmission in a community since OPV vaccinations were discontinued in 2000 (2–4). The extent of circulation within the affected community is not yet known. However, the identification of poliovirus infection in the index patient and three other unvaccinated children in a community at high risk for poliovirus transmission raises concerns regarding 1) transmission to other communities with low levels of vaccination and 2) the risk for a polio outbreak occurring in the United States. Potential also exists for transmission of this virus to other immunodeficient persons. Although this VDPV has not been associated with paralytic disease, based on previous experience with VDPVs, the virus is considered to have potential both for wider transmission and for causing paralytic disease.

VDPVs emerge from OPV viruses as a result of 1) their continuous replication in immunodeficient persons (immunodeficiency-associated or iVDPVs) such as the index patient

in this investigation or 2) their circulation in populations with low vaccination coverage (circulating or cVDPVs) (1). During community circulation, cVDPVs often recombine with other species C enteroviruses, which is not characteristic for iVDPVs (1). Because polioviruses accumulate nucleotide changes at a constant rate of mutation (approximately 1% per year), the time of replication can be inferred from the degree of divergence (1). Because cVDPVs commonly revert to a wild poliovirus phenotype, they can have increased transmissibility and high risk for paralytic disease; cVDPVs have caused outbreaks of poliomyelitis in several countries (1). VDPVs in highly immunized populations are rare. Before the VDPV identification in Minnesota, the most recent known VDPV excreter in the United States was a child with SCID (now deceased) who developed vaccine-associated paralytic poliomyelitis in 1995 (4).

Given the degree of difference (2.3%) from the parent Sabin poliovirus type 1 strain, the virus isolated from the index patient is estimated to have been replicating for approximately 2 years, which means the virus likely is older than the infant. OPV is still widely used in most countries; however, because OPV has not been used in the United States since 2000 and in Canada since 1997, the original source of this virus likely was a person who received OPV in another country. Neither the infant nor her family members had any history of international travel. This virus is not related to other known iVDPVs or to any type 1 cVDPVs that caused outbreaks such as those in Hispaniola during 2000–2001, the Philippines during 2001 (1), or Indonesia during 2005.

Most poliovirus infections are asymptomatic or cause mild, febrile disease. Poliovirus infections occasionally cause aseptic meningitis and one out of 200 infections from poliovirus type 1 results in paralytic poliomyelitis, characterized by acute onset of flaccid paralysis that is typically asymmetric and associated with a prodromal fever. Poliovirus is spread through fecal material, oral secretions, and fomites. Widespread transmission among vaccinated health-care workers or in a community with high vaccination coverage is unlikely because fully vaccinated persons are not at risk for disease from this or other polioviruses and seldom shed the virus for longer than a week if they are infected. The National Immunization Survey reports that polio vaccination coverage in Minnesota is 93% for children aged 19–35 months and 98% for school-aged children; however, communities of unvaccinated persons exist in Minnesota and many other states (5). The risk for transmission in communities with low vaccination coverage is high. The estimated rate of transmission for wild poliovirus among unvaccinated household contacts is 73%–96% (6). Contacts between persons in communities with low vaccination coverage pose the potential for transmission of this poliovirus to

other communities in the United States, Canada, and other countries.

The last wild poliovirus outbreak in the United States occurred in 1979 and was caused by a wild type 1 poliovirus. In that outbreak, 10 paralytic poliomyelitis cases and four other poliovirus infections occurred among unvaccinated Amish persons and members of other religious communities with low levels of vaccination who lived in Iowa, Missouri, Pennsylvania, and Wisconsin. The source of this outbreak was traced to religious groups in Canada and the Netherlands that also had low levels of vaccination (7). A polio outbreak in 1993 in the Netherlands with 71 paralytic cases among members of unvaccinated religious communities also resulted in poliovirus transmission without paralytic disease in Alberta, Canada; no evidence of transmission from this outbreak was found in the United States (8).

Persons in communities with low vaccination coverage should be warned of the potential risk for poliomyelitis. States with large communities with low vaccination coverage should identify these communities, assess their current vaccination status, and offer IPV. These states also should establish enhanced or active surveillance for AFP, GBS, and transverse myelitis. Physicians should be aware of and vigilant for poliomyelitis and other causes of AFP in patients. Stool samples, throat swabs, cerebrospinal fluid, and serum should be collected for viral culture and serology from these patients. With evidence of transmission in Minnesota, serologic and/or stool surveys to detect poliovirus type 1 circulation in affiliated communities with low levels of vaccination also should be considered.

IPV, the polio vaccine currently used in the United States, provides immunity against this vaccine-derived poliovirus strain. The Advisory Committee on Immunization Practices (ACIP) recommends that a full 3-dose IPV series be administered on an accelerated schedule if polio immunization status is unknown or not documented (9). A booster dose of IPV is recommended for adults in susceptible communities and health-care workers at high risk for exposure who have completed a primary series but have not received an adult booster dose.

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