06-ID-15

Committee: Infectious Diseases

Title: Inclusion of Poliovirus Infection (Paralytic and Nonparalytic) Reporting in the National Notifiable Diseases Surveillance System

Statement of the Problem

In September 2005, a type 1 vaccine-derived poliovirus (VDPV) was identified in a stool specimen obtained from an Amish infant undergoing evaluation for recurrent infections, diarrhea and failure to thrive, and subsequently diagnosed as having an immune deficiency disorder (1). Hospital investigations did not find a healthcare-associated source for the infant's infection or evidence of nosocomial transmission from the infant to other patients or healthcare workers. Investigation of the infant's central Minnesota community identified 4 other children in 2 other households infected with the VDPV, and serologic tests indicated that 3 of the infant's siblings had likely been recently infected with a type 1 poliovirus. Analysis of genomic sequence data from the poliovirus isolates suggested that the VDPV had been introduced into the community about 3 months before the infant was identified. Together, the data suggested that there had been circulation of the virus in the small community. Investigations in other communities in Minnesota and nearby states and Canada did not identify any additional infections and no cases of paralytic poliomyelitis were detected.

Although oral poliovirus vaccine (OPV) is still widely used in most countries, IPV replaced OPV in the United States in 2000 (2). Therefore, the Minnesota VDPV infections were 1) the result of importation of a vaccine-derived poliovirus into the United States, and 2) the first documented occurrence of VDPV community transmission in a developed country (3-4). Circulating VDPVs have recently caused polio infections and outbreaks of paralytic poliomyelitis in several countries (3-5). Because circulating VDPVs commonly revert to a wild poliovirus phenotype, they can have increased transmissibility and high risk for paralytic disease (3). Contacts between persons in communities with low polio vaccination coverage pose the potential for transmission of poliovirus and outbreaks of paralytic poliomyelitis.

In the United States, the last wild poliovirus outbreak occurred in 1979; the last imported wild poliovirus case occurred in 1993; and the last vaccine-associated paralytic poliomyelitis (VAPP) case occurred in 1997, with the vaccine policy change from OPV to all IPV. In the Americas, the last indigenously-acquired wild poliovirus case occurred in Peru in 1991 due to the success of the Polio Eradication Initiative. With the elimination of polio in the region of the Americas, the occurrence of any poliovirus infections in the U.S. is a cause for concern. Reflecting the global concern for poliovirus importations into previously polio-free countries, the World Health Assembly, W.H.O., has added circulating poliovirus to the notifiable events in the International Health Regulations (IHR) (6).

Statement of the desired action(s) to be taken:

1. Poliovirus (PV) infection (paralytic and nonparalytic infections) should be nationally reportable and reported through the National Notifiable Diseases Surveillance System.
2. When a poliovirus infection is identified, state health officials and the CDC should be notified immediately for confirmatory virus testing, if needed, and case and contact investigation.
3. If a poliovirus infection is identified in a community with low polio vaccination coverage (e.g., IPV3 coverage less than 80% in 19-35 month old infants), active surveillance for AFP, Guillan-Barre Syndrome (GBS), and transverse myelitis should be implemented in the affected community and contact communities. Stool samples, throat swabs, and cerebrospinal fluid (if clinically indicated) for viral culture and serum for serologic testing should be collected from contacts of infected persons and persons with illnesses clinically compatible with poliovirus infection, especially in low-coverage communities.

Goals of Reporting and Surveillance:

1. Increase awareness among physicians to be aware of and vigilant for poliomyelitis and other causes of acute flaccid paralysis (AFP) in patients.
2. Document the detection of PV infections – both wild and vaccine-derived.
3. After such detections, establish more sensitive surveillance (AFP reporting and testing for PV) and increase vaccination coverage in communities that refuse vaccination.

Methods for Reporting and Surveillance:

1. Sources of surveillance data for PV infection include public health and private laboratories that do enterovirus testing and virus identification, physicians and other health care providers, and public health authorities.
2. When a poliovirus infection is identified, state health officials and the CDC should be notified immediately for confirmatory virus testing, if needed, and case and contact investigation.
3. If a poliovirus infection is identified in a community with low polio vaccination coverage (e.g., IPV3 coverage less than 80% in 19-35 month old infants), active surveillance for AFP, Guillan-Barre Syndrome (GBS), and transverse myelitis should be implemented in the affected community and contact communities. Stool samples, throat swabs, and cerebrospinal fluid (if clinically indicated) for viral culture and serum for serologic testing should be collected from contacts of infected persons and persons with illnesses clinically compatible with poliovirus infection, especially in low-coverage communities.

Case Definition Narrative:

Clinical presentation: 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, some aerosols and fomites.

Laboratory evidence: Polioviruses are among the most rapidly evolving of all RNA viruses (1). During community circulation, cVDPVs often recombine with other species C enteroviruses (3). 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 (3). Poliovirus isolates are characterized according to their genetic properties and all vaccine-related poliovirus isolates should be evaluated by genomic sequencing to determine degree of divergence from the parent Sabin strains (3). In particular, sequencing should be performed on the virus capsid protein coding region (VP1) of the poliovirus genome to identify the virus as VDPV, and analysis of recombination with other polioviruses or species C enteroviruses should be determined.

Criteria for epidemiologic linkage: Consider vaccination coverage (community), vaccination history (individual), immunocompetence, symptoms or lack thereof, and poliovirus exposure risks (e.g. travel).

Case Definition

Confirmed:	Poliovirus isolate identified in appropriate clinical specimen, with confirmatory typing and sequencing performed by the CDC Poliovirus Laboratory as needed.
Probable:	none
Suspect:	none

Period of Surveillance: Ongoing

Background and Justification:

Oral poliovirus vaccine (OPV) is still widely used in most countries; however, OPV has not been used in the United States since 2000 nor in Canada since 1997. Vaccine-derived polioviruses (VDPVs) are poliovirus strains derived from one of the three Sabin poliovirus strains in oral polio vaccine (OPV) that have ≥1% difference in nucleotide sequence in the viral capsid protein 1 region from the prototype vaccine virus (3-4). VDPVs emerge from OPV viruses as a result of 1) the OPV virus' continuous replication in immunodeficient persons (immunodeficiency-associated or iVDPVs), or 2) the OPV circulation in populations with low vaccination coverage (circulating or cVDPVs). Vaccine-derived polioviruses have recently caused polio infections and outbreaks of paralytic poliomyelitis in several countries (3-6). During 2005, public health officials made the first identification of VDPV transmission in a community since OPV vaccinations were discontinued in 2000 (3-6).

Because cVDPVs commonly revert to a wild poliovirus phenotype, they can have increased transmissibility and high risk for paralytic disease (3-4). VDPVs in highly immunized populations are rare, and widespread transmission among vaccinated health-care workers or in a community with high vaccination coverage is unlikely. However, communities of unvaccinated persons exist in many states (7). The risk for transmission in communities with low vaccination coverage is high (11). The estimated rate of transmission for wild poliovirus among unvaccinated household contacts is 73–96% (2). The most significant risk factor for cVPDV outbreaks, like wild poliovirus outbreaks, is insufficient population immunity (3-4). 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.

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