Communications

Duration of Vaccine-Induced Poliomyelitis Immunity

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The introduction of poliovirus vaccines in 1955 dramatically reduced the number of cases of poliomyelitis. Small outbreaks still occur where immunization levels are low, and a few cases of vaccine-related poliomyelitis develop in nonimmune adults exposed to recently immunized children. To be effective, an immunization program must protect these groups as well.

Vaccination policies for preventing poliomyelitis have not changed since a supplementary booster of oral polio vaccine (OPV) at five years of age was recommended. This supplementary booster was added because serologic data demonstrated that a small number of children do not produce antibodies after the primary series of three immunizations.[1]

In 1977 a special committee investigating poliovirus recommended an additional OPV booster for entrants of the seventh grade of school or its equivalent.[2] This recommendation has been disregarded, as it is currently felt that the need for supplementary immunizations, after the primary series and a booster upon entering school, has not been established.[3]

Serologic data on the polioimmune status of entrants to the seventh grade (aged approximately 12 or 13 years) who have received OPV as recommended, however, are either lacking or have come from atypical populations, such as military dependents or institutionalized children.[4,5] Such serologic data would be useful in determining the need for supplementary OPV boosters. In addition, such data would help to settle the controversy regarding the use of inactivated polio vaccine (IPV), which is safer than OPV, but does require boosters every ten years.[2]

The lack of representative serologic data prompted this study of antibody titers to poliovirus in 12- to 13-year-old children who had documented immunizations with OPV exclusively and according to currently recommended schedules.

Methods

Study Population

A computer search of medical records from the Park Nicollet Medical Center identified 225 patients, 12 or 13 years of age, who had been born and had received their immunizations through the clinic. These individuals were informed of the study and its purpose by mail and asked to participate. An additional two mailings were required to obtain voluntary agreement from 63 individuals to take part in the study, a response rate of 28 percent.

Of the 63 adolescents in the study population, 27 were aged 12 years and 36 were aged 13 years. Their clinic records documented immunization with trivalent oral polio vaccine. Fifty-two had been immunized according to the three-dose primary series recommended by the US Public Health Service Advisory Committee on Immunization Practices (ACIP) and 11 were immunized according to the four-dose primary series recommended by the Committee on Infectious Diseases of the American Academy of Pediatrics. In addition, all had received a booster of OPV at between 4 and 6 years of age upon entering school. The last immunization prior to testing had been 6 years ago for 6 of the participants, 7 years ago for 30, 8 years ago for 25, and 9 years ago for 2.

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Serology

The microneutralization system used poliovirus types 1, 2, and 3, titrated and used at a concentration of 100 tissue culture ID_{50}/0.1 mL, plus control antisera to poliovirus types 1, 2, and 3, both obtained from American Type Culture Collection. The control and test antisera were both heat inactivated at 56° C for 30 minutes before testing. Control antisera containing 50 antibody U/0.1 mL and test sera at a dilution of 1:2 were then incubated with the specific polioviruses at 37° C for 90 minutes. Then 50 μL of the virus-serum mixtures were inoculated into 96 well-plates of primary cynomolgus monkey kidney cell cultures. These cultures were incubated at 37° C with 5 percent carbon dioxide and observed daily for cytopathic effect. Incubation was terminated and results determined when virus controls contained a 4+ cytopathic effect. Sera were considered positive for a specific antibody if development of cytopathic effect was inhibited in at least one well.

Sera that were negative in the microneutralization assay were retested by a plaque reduction technique. Poliovirus types 1, 2, and 3 were titrated and 20 to 25 plaque-forming units of each type were used in tests. Virus-serum mixtures were incubated for one hour at 37° C and then inoculated into 12 well-plates of primary cynomolgus monkey kidney cells. The cells were then overlaid with 1 percent agarose in minimal essential medium with 4 percent fetal bovine serum. Two days later a second overlay of 1 percent agarose in minimal essential medium with 0.005 percent neutral red was added. Plaques were counted 12 hours later, and the results expressed as the mean number of plaques for triplicate wells. Sera were considered positive if they reduced the number of plaques at least 80 percent compared with the control virus titration incubated in the absence of antisera.

Results

When the 63 sera were assayed by the microneutralization system for antibodies to poliovirus types 1, 2, and 3 at a dilution of 1:2, only one specimen was found to lack antibody. This serum lacked detectable antibody to poliovirus type 1; however, when it was retested by the plaque-

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reduction technique, it did demonstrate the presence of antibody to poliovirus type 1. The subject involved was 12 years old, had been immunized by the three-dose ACIP schedule, and had received a booster seven years previously. Review of this subject's past medical history did not reveal any apparent immunologic disease.

Comment

It has been shown previously that neutralizing antibody to poliovirus declines over time and can be boosted with an additional dose of OPV.[4] This study, however, demonstrates that adequate immunity was obtained from the present immunization policies for poliomyelitis and persisted at least six to nine years. Another booster at 12 years of age would therefore not offer increased protection at that age.

An additional booster at 12 years of age might, however, prolong the protection that exists at that time. This prolonged protection could eventually reduce the number of adults who because of their own inadequate immunity are at risk for contracting poliomyelitis from immunized infants.[6] It is not known whether lack of immunity in this adult group results from inadequate immunization or declining antibody titers.[7] Repeating this study on the same population in subsequent years may provide useful information in this regard.

Answering these questions about the duration of polio immunity is essential if a safe and effective national polio immunization policy is to be established. If polio immunity does indeed decline over time and additional boosters are needed, perhaps on a regular basis, then a switch to IPV might be indicated for the program as a whole. IPV is a safer vaccine than OPV, its only disadvantage being the need for regular boosters.

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An Extended Family With Giardiasis

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Asymptomatic infants with giardiasis may be the unsuspected cause of treatment failure in other extended-family members. The family physician must maintain a very high index of suspicion for this problem. Giardiasis is often asymptomatic,[1] but little doubt remains that the organism can produce symptoms.[2] Waterborne,[3] foodborne,[4] and person-to-person[5] transmission of Giardia lamblia has been observed. Galazka points out that the family physician must consider the source whenever giardiasis is identified in an individual.[6] This case report describes individuals whose giardiasis was not cured until members of the extended fam-

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