Varicella vaccine update: Need for a booster?

Background and epidemiology: Between Dec. 1, 2000, and Jan. 11, 2001, 25 (28%) of 88 children at a daycare centre in New Hampshire came down with chickenpox.[1] Perhaps not a surprising finding in the pre-vaccine era, but in this outbreak 73% of the children old enough to receive the vaccine had been vaccinated. The index case was a healthy 4-year-old boy who had been vaccinated 3 years previously. This outbreak raises new concerns over the effectiveness of the varicella vaccine and whether a booster dose is needed.[2]

In the United States the varicella vaccine was approved in 1995 by the US Food and Drug Administration. By 2000 more than 75% of young American children had been vaccinated and the incidence of varicella and varicella-related hospital admissions had declined by 80%.[3]

The vaccine was licensed in Canada in 1998[4] and its use recommended by the National Advisory Committee on Immunization in 1999[5] and the Canadian Task Force on Preventive Health Care in 2001.[6] Data for Canada are patchy; however, it is likely that the vaccine is not widely used and that the incidence of varicella and related complications requiring hospital admission remains unchanged (Dr. Arlene King, Director, Immunization and Respiratory Diseases, Centre for Infectious Disease Prevention and Control, Health Canada: personal communication, 2002). The vaccine, its use and characteristics were reviewed in this column about a year ago.[7]

Single-dose vaccination is recommended for children 12–15 months of age. The vaccine can be given simultaneously with the measles–mumps–rubella (MMR) vaccine at a separate site. Catch-up vaccination, again in a single dose, is recommended for susceptible children (those who have not already had chickenpox) between 15 months and 12 years of age. For older susceptible children and adults, 2 doses 4–8 weeks apart are recommended.

Clinical management: The effectiveness of the varicella vaccine is estimated to be between 70% and 90%.[7] In the New Hampshire outbreak, it was 44%.

Reasons for vaccine failure can be primary or secondary. Primary failure occurs when the vaccination does not seem to "take." The main reasons for this are improper handling and storage of the vaccine. Other factors related to primary failure of the varicella vaccine are a history of asthma, lower age at vaccination, and simultaneous or closely spaced varicella and MMR vaccine administration,[2] although none of these was a risk factor in the New Hampshire outbreak. Higher doses of vaccine may provide better protection.[7] Secondary failure is due to waning immunity with the passage of time.

Fourteen days is the typical incubation period for wild-virus varicella infection; therefore, rashes that occur within 14 days after vaccination are almost always the result of infection that occurred before vaccination. Between 14 and 42 days after vaccination, the typical rash may be due to the wild virus or to the vaccine virus; the distinction can be made only through molecular typing. Cases of varicella that occur in vaccinated people more than 42 days after vaccination are referred to as "breakthrough cases" and are due to the wild virus.

Compared with naturally occurring varicella in nonvaccinated people, breakthrough disease is milder and results in fewer lesions (usually fewer than 50) and fewer complications.

Prevention and control: The implications of such a high degree of vaccine failure are 3-fold. First, although vaccinated children experiencing breakthrough varicella will be less ill than nonvaccinated children with naturally occurring chickenpox, they are still capable of transmitting the wild virus and causing outbreaks, as was the case in New Hampshire. One of the great benefits of mass vaccination is the virtual elimination of natural reservoirs of the wild virus.

Second, it is believed that the vaccine virus is much less likely than the wild virus to result in secondary infection (herpes zoster).[2] Breakthrough disease presumably negates this benefit by exposing the vaccinated person to the wild virus and thus increasing the chance that zoster will develop.

Finally, maternal infection with wild varicella zoster virus during the first 28 weeks' gestation can result in transmission of the virus to the fetus and lead to congenital varicella syndrome.[8] Presumably, vaccinated women who experience breakthrough disease would also expose their children to congenital varicella syndrome.

Physicians and public health practitioners should continue their current practices and follow the recommendations of expert groups. However, these groups will need to rethink varicella vaccine strategies. Also, surveillance of varicella infection should be heightened in both vaccinated and nonvaccinated populations and vaccine coverage rates monitored.

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References

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