Resurgence of *Bordetella pertussis* infection

Epidemiology: Seventeen pertussis-related deaths were reported in the United States in 2000.[1] All involved infants less than 4 months old who were too young to be fully protected by vaccination. As tragic as these deaths were, from a population health perspective the small numbers marked a paradoxical victory for current national vaccination programs. Compare those 17 deaths with data from 1934, before the introduction of routine vaccination, when more than 12 000 children died of pertussis in the United States.[2] Today death tolls of more than 350 000 annually persist in poorer nations that are unable to sustain effective vaccination programs.[3] In rich countries, thanks to government-supported vaccination programs, we can now speak of pertussis in terms of morbidity, not mortality.

Unfortunately, morbidity trends in Canada, and in developed countries in general, are not so good. Since the early 1990s there has been a resurgence in pertussis activity despite high vaccine coverage[4,5] — due, perhaps, to incomplete vaccination, waning immunity from a poorly protective old vaccine, or increased awareness and detection of pertussis in older people. Whereas children less than 10 years of age are by far the most affected group, both the number and proportion of cases involving older people have increased over the last decade, triggering renewed interest in the pathogenesis of pertussis and prompting the question: Are 10-year pertussis boosters indicated?[4]

Bordetella pertussis is a small, gram-negative rod that causes severe respiratory disease in humans. It has no known animal or environmental reservoir; humans are the only natural host and assumed reservoir. Outbreaks occur every 2–5 years, mostly in the summer and early fall, even in populations with high vaccine coverage. This suggests that, although vaccination appears to control disease, it has little influence on transmission.[6] A number of virulent gene factors, such as pertussis toxin, filamentous hemagglutinin, pertactin, fimbrial agglutinogens and adenylate cyclase, play a role in the pathogenesis and protective immunity of pertussis infection.[3,6] The incubation period is typically 5–7 days.

Clinical management: Pertussis typically lasts several weeks and has 3 stages: catarrhal (rhinorrhea and mild cough), paroxysmal (with increasing severity of cough and repetitive coughing spells, followed by an inspiratory whoop or posttussive vomiting, or both) and convalescent (decreasing severity and frequency of coughing spells).[6] Infants are most susceptible to serious complications, including seizures, encephalopathy, secondary bacterial pneumonia, apnea and pulmonary hypertension.[1,3,6] The spectrum of symptoms is usually less severe in older children and adults, in whom pertussis is an often unrecognized cause of chronic cough or respiratory illness.

Pertussis is a reportable disease, and suspected cases of paroxysmal, prolonged cough should be investigated and reported to the local public health office. Isolation of the organism from nasopharyngeal secretions is considered the "gold standard" for diagnosis. Serologic testing for significant rises in antibody titres and polymerase chain reaction are additional diagnostic methods. Antibiotic treatment will shorten the course of disease, especially if given in the catarrhal phase. A 2-week course of erythromycin is the treatment of choice,[7] although the parents of infected infants should be informed about the possible risk of pyloric stenosis.[3] Clarithromycin, azithromycin or trimethoprim–sulfamethoxazole are possible alternatives.[3,7]

Prevention: Prevention is possible through avoidance of exposure, prophylactic chemotherapy and vaccination. Adults are the primary source of pertussis for infants in hospital, so adults with a new-onset, persistent, not-yet-diagnosed cough should try to stay away from infants. A 14-day course of erythromycin for household and other close contacts, regardless of immunization status and age, is recommended.

Acellular pertussis vaccines, containing pertussis toxoid, filamentous hemagglutin and pertactin, have replaced older, whole-cell pertussis vaccines in Canada. Data suggest the acellular pertussis vaccines have an estimated efficacy of about 85% and carry significantly lower rates of adverse reactions (i.e., mild local and general reactions, extremely rare persistent crying or hypotonic-hyporesponsive episodes) than the older whole-cell vaccine. Immunization routinely consists of 3 doses given at 2, 4 and 6 months, followed by a fourth at 18 months and a fifth at 4 to 6 years. A combined diphtheria-tetanus, acellular pertussis (dTap) booster dose for adolescents and adults has been licensed and should be used to replace the adolescent booster of Td. Until data about the safety of repeated doses are available, only one dose is currently recommended.[8]

Erica Weir
CMAJ

References

1. Pertussis Deaths – United States, 2000. *MMWR Morbid Mortal Wkly Rep* 2002;51(28):616-8. Available: www.cdc.gov/mmwr (accessed 2002 Sept 26).
2. Klein D. From pertussis to tuberculosis: What can be learned? *Clin Infect Dis* 2000;30(Suppl 3):S302-8.
3. Schleiss M, Dahl K. Acelluar pertussis vaccines. *Curr Probl Pediatr* 2000;July:185-201.
4. Orenstein W. Pertussis in adults: epidemiology, signs, symptoms and implications for vaccination. *Clin Infect Dis* 1999;28(Suppl 2):S147-50.
5. De Serres G. An Advirosy Committee Statement (ASC). National Advisory Committee on Immunization (NACI). Statement on adult/adolescent formulation of combined acellular pertussis, tetanus, and diphtheria vaccine. *Can Commun Dis Rep* 2000;26:1-8.
6. Heininger U. Recent progress in clinical and basic pertussis research. *Eur J Pediatr* 2001;160: 203-13.
7. Kerr JR, Matthews RC. *Bordetella pertussis* infection: pathogenesis, diagnosis, management, and the role of protective immunity. *Eur J Clin Microbiol Infect Dis* 2000;19:77-88.
8. *Canadian Immunization Guide 2002.* 6th ed. Ottawa: Health Canada; 2002.