Pertussis

General consideration

Pertussis is an acute respiratory tract infection that was well described initially in the 1500s. Sydenham first used the term pertussis, meaning "intense cough", in 1670; it is preferable to whooping cough because most infected individuals do not “whoop.”

Pertussis is an acute, highly communicable infection of the respiratory tract caused by *Bordetella pertussis* (gram negative fastidious coccobacillus) and characterized by severe bronchitis. Transmission occurs by close contact with cases via aerosolized droplets. Children usually acquire the disease from symptomatic family contacts. Adults who have mild respiratory illness, not recognized as pertussis, frequently are the source of infection. Asymptomatic carriage of *B. pertussis* is not recognized. Infectivity is greatest during the catarrhal and early paroxysmal cough stage (for about 4 weeks after onset).

B. pertussis organisms attach to the ciliated respiratory epithelium and multiply there; deeper invasion does not occur. Disease is due to several bacterial toxins, the most potent of which is "pertussis toxin", which is responsible for lymphocytosis and many of the symptoms of pertussis.

Bordetella parapertussis causes a similar but milder syndrome.

Pertussis is extremely contagious, with attack rates as high as 100% in susceptible individuals exposed to aerosol droplets at close range.

Neither natural disease nor vaccination provides complete or lifelong immunity against pertussis re-infection or disease. Protection against typical disease begins to wane 3-5 yr after vaccination.

Coughing adolescents and adults (usually not recognized as having pertussis) are the major reservoir for *B. pertussis* and are the usual sources of infection for infants and children.

CLINICAL MANIFESTATIONS

An incubation period ranging from 3–12 days. Pertussis is a prolonged disease that

usually divided into 3 stages:-

1. **Catarrhal stage** (1–2 wk) begins insidiously as flu-like illness e.g. low- grade fever, rhinorrhea, sneezing, and lacrimation.

2. **Paroxysmal stage** (2–6 wk), the cough begins as a dry, intermittent, irritative hack that evolves into a machine-gun burst of uninterrupted coughs which followed by whoop, exhaustion, & vomiting (post-tussive emesis); at the peak of the paroxysmal stage there may be >1 episode/hr.

3. **Convalescent stage** (≥2 wk) begins when the number, severity, and duration of episodes diminish.

Infants < 3 mo do not display classical stages; the catarrhal phase lasts only a few days or is unnoticed; cough may not be prominent or manifests as expiratory grunt; whoop infrequently occurs in infants <3 mo; whereas cyanosis can follow a coughing paroxysm.

Apnea may be the only symptom & can occur without cough. The paroxysmal and convalescent stages in young infants are lengthy.

Paradoxically, in infants the cough and whooping may become louder and more classic in the convalescent stage. Exacerbation of cough may recur after subsequent RTI.

Findings on physical exam are generally uninformative. Signs of lower respiratory tract disease are not expected unless complicating secondary bacterial pneumonia is present.

Note: *Pertussis should be suspected in any individual who has pure or predominant complaint of cough for ≥2 wk duration with at least 1 associated symptoms of either: paroxysms, whoop, or post-tussive emesis*

DIAGNOSIS

1. **CBP**: Leukocytosis (15,000–100,000 cells/mm3) is due to **absolute lymphocytosis** (which is the characteristic of **catarrhal stage**); it is due to normal T- & B-cells. Extreme leukocytosis and thrombocytosis has been correlated with severe course of disease and death.
2. **CXR** only **mildly abnormal** e.g. perihilar infiltrate or edema & atelectasis. Parenchymal consolidation suggests secondary bacterial infection.
3. **Culture of *B. pertussis*** remains the gold standard for Dx, but careful attention must be directed to specimen collection, transport, and isolation. Specimen is obtained by deep nasopharyngeal aspiration or by use of swab held in the posterior nasopharynx for 15–30 sec (or until coughing). *Regan-Lowe charcoal agar* is the preferred media for these fastidious organisms.
4. **Direct Fluorescent Antibody (DFA) & PCR** of potential isolates are rapid tests that maximize recovery.
   Note: *The culture, DFA & PCR are usually +ve during the catarrhal and early paroxysmal stages.*
5. **Serology**: 2 folds ↑ of IgG to the PT indicate recent infection.
6. **Blood glucose** may show hypoglycemia (due to mild hyperinsulinemia).

Differential diagnosis

1. *Bordetella parapertussis* and *Bordetella bronchiseptica*.

2. Mycoplasma pneumonia

3. *Chlamydia trachomatis* and *Chlamydophila pneumonia*

4. Respiratory tract viruses, particularly *adenoviruses* and *respiratory syncytial viruses*.

COMPLICATIONS

Pertussis has many Cxs which mainly affect the young infants e.g.

pneumonia (25%), **seizures** (4%), **encephalopathy** (1%), and **death**

1. **Respiratory failure** may occur due to apnea or secondary bacterial pneumonia, especially if associated with pulmonary hypertension or hemorrhage. Secondary bacterial pneumonia is mainly caused by *Staphylococcus aureus, Streptococcus pneumonia* or "oropharyngeal flora".
2. **Seizures** may be due to hypoxemia, intracranial hemorrhage, or hyponatremia (due to SIADH); whereas PT donot cause seizure.
3. **Sequelae of ↑ intrathoracic and intra-abdominal pressure** during coughing include: conjunctival, scleral, retinal, and intracranial hemorrhages, petechiae on the upper body, epistaxis, pneumothorax, and subcutaneous emphysema, umbilical and inguinal hernias as well as laceration of the lingual frenulum.
4. **Death** has been associated with hx of prematurity & young maternal age. Pertussis may cause SIDS.

TREATMENT

Indicatios of admission to the hospital include: ***infants below 3 mo*** (especially when there is hx of prematurity) & ***infants above 3 mo*** when there is **underlying** diseases, **Cxs** (as above) or **severe paroxysm** that characterized by: duration >45 sec; cyanosis; bradycardia; hypoxia; paroxysm is not end by whoop or strength for self-rescue or cannot expectorate mucus plug; and post-tussive unresponsiveness.

 The infant should have **intubation, paralysis, and ventilation** if there is hx of respiratory failure, repeated apnea, or life-threatening events.

 Antibiotics should always be given when pertussis is suspected or confirmed primarily to limit the spread of infection and secondarily for possible clinical benefit.

Macrolides are the preferred agents e.g. **Azithromycin**, 10 mg/kg once daily for 5 days (for infants <6 mo) & 10 mg/kg in 1st day then 5 mg/kg for subsequent 4 days (for infants >6 mo & children), **Erythromycin** 40- 50 mg/kg ÷ 4 for 2 wk; or **Clarithromycin** 15 mg/kg ÷ 2 for 1 wk.

Note: *Erythromycin & Clarithromycin should not be given to infant below 1 mo of age due to the risk of pyloric stenosis. TMP-SMZ is an alternative agent to macrolide but it also contraindicated in infants below 2 mo.*

 Adjunctive therapy. Patients should be nursed in a quiet, dimly light room with **oxygen & suction** (but it may provoke the paroxysm); **mist** by tent may ameliorate thick, tenacious secretions. Feeding between paroxysms is important but avoid over feeding. Corticosteroids & bronchodilators e.g. β-agonists are of controversial use in pertussis.

All patients with pertussis (including suspected cases) should be placed in a room with respiratory isolation with use of mask by all health personnel who enter the room till 5 days of initiation of macrolide Rx. Macrolide Px should also be given to all household or any close contacts regardless of age, symptoms or immunization status. After discharge from hospital, family education & support are important measures.

Close contacts should also be vaccinated according to their age as follow: Children <7 yr who have received <4 doses of pertussis- containing vaccines should have vaccination initiated or continued to complete the recommended series, whereas those who receive 3rd dose >6 mo or 4th dose ≥3 yr before exposure should receive a booster dose. Individuals ≥9 yr should be given Tdap vaccine.

Note: *Protection against typical disease begins to wane after 3–5 yr of vaccination, and is unmeasurable after 12 yr. Mothers provide little (if any) passive protection to young infants during pregnancy & breast feeding.*

PREVENTION

DTaP (acellular pertussis) has replaced DPT (whole-cell pertussis)

vaccine to minimize the SE e.g. high fever, persistent crying of ≥3 hr, hypotonic hyporesponsive episodes, & seizures.

Schedule of immunization is at 2, 4 & 6 mo of life. 1st booster is at 15-18 mo & the 2nd booster at 4-6 yr of life. Adolescents >11 yr & adults can be given Tdap.

Prognosis

The prognosis for patients with pertussis has improved in recent years because of excellent nursing care, treatment of complications, attention to nutrition, and modern intensive care. However, the disease is still very serious in infants under age 1 year (esp <3 mo); most deaths occur in this age group. Children with encephalopathy have a poor prognosis. Case-fatality rates are approximately 1% in infants younger than 2 months of age.