Pertussis (whooping cough) *Professor Dr.Dawood Al-Azzawi

(Bordetella Pertussis & Bordetella Parapertussis)

Pertussis is an acute respiratory tract infection described initially in the 1500s.

Etiology;

Bordetella pertussis is the sole cause of epidemic pertussis and the usual cause of sporadic pertussis. Bordetella parapertussis is an occasional cause of sporadic pertussis B. pertussis and B. parapertussis are exclusive pathogens of humans and some primates. Protracted coughing can be caused by Mycoplasma, parainfluenza or influenza viruses, enteroviruses, respiratory syncytial virus, or adenoviruses.

Epidemiology;

Before vaccination was available, pertussis was the leading cause of death due to communicable disease among children <14 yr of age in the United States, with 10,000 deaths annually. Widespread use of pertussis vaccine led to a >99% decline in cases. Pertussis is increasingly endemic, with less cycling or seasonality than previously occurred..

Pertussis is extremely contagious, with attack rates as high as 100% in susceptible individuals exposed to aerosol droplets at close range. *B. pertussis* does not survive for prolonged periods in the environment. Chronic carriage by humans is not documented. Neither natural disease nor vaccination provides complete or lifelong immunity against reinfection or disease. Coughing adolescents and adults (usually not recognized as having pertussis) currently are the major reservoir for *B. pertussis* and are the usual sources for "index cases" in infants and children.

Pathogenesis;Bordetella organisms are gram-negative coccobacilli that only colonize ciliated epithelium. Exact mechanism of disease symptomatology remains unknown.

Clinical manifestations.

Classically, pertussis is a prolonged disease, divided into catarrhal, paroxysmal, and convalescent stages. The **catarrhal stage** (1–2 wk) begins insidiously after an incubation period ranging from 3–12 days with nondistinctive symptoms of congestion and rhinorrhea variably accompanied by low-grade fever, sneezing, lacrimation, and conjunctival suffusion. As initial symptoms wane, coughing marks the onset of the **paroxysmal stage** (2–6 wk). The cough begins as a dry, intermittent, irritative hack and evolves into the inexorable paroxysms that are the hallmark of pertussis. A well-appearing, playful toddler with insignificant provocation suddenly expresses an anxious aura and may clutch a parent or comforting adult before beginning a machine-gun burst of uninterrupted coughs, chin and chest held forward, tongue protruding maximally, eyes bulging and watering, face purple, until coughing ceases and a loud whoop follows as inspired air traverses the still partially closed airway. Post-tussive emesis is common, and exhaustion is universal. The number and severity of paroxysms escalate over days to a week and remain at that plateau for days to weeks. At the peak of the paroxysmal stage, patients may have more than 1 episode hourly. As the paroxysmal stage fades into the **convalescent stage** (≥2 wk), the number, severity, and duration of episodes diminish

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Infants >3 mo of age do not display classical stages. Whoop infrequently occurs in infants <3 mo of age who at the end of a paroxysm lack stature or muscular strength to create sudden negative intrathoracic pressure. Cyanosis can follow a coughing paroxysm, or apnea can occur without a cough. Apnea may be the only symptom. The paroxysmal and convalescent stages in young infants are lengthy. Findings on physical examination generally are uninformative. Signs of lower respiratory tract disease are not expected unless complicating secondary bacterial pneumonia is present. Conjunctival hemorrhages and petechiae on the upper body are common.

Diagnosis and differential diagnosis;

Pertussis should be suspected in any individual who has pure or predominant complaint of cough, especially if the following are absent: fever, malaise or myalgia, exanthem or enanthem, sore throat, hoarseness, tachypnea, wheezes, and rales. For sporadic cases, a clinical case definition of cough of ≥14 days' duration with at least 1 associated symptom of paroxysms, whoop, or post-tussive vomiting has a sensitivity of 81% and specificity of 58% for culture confirmation. Pertussis should be suspected in older children whose cough illness is escalating at 7–10 days and whose coughing episodes are not continuous. Pertussis should be suspected in infants <3 mo of age with apnea, cyanosis, or an acute life-threatening event (ALTE). B. pertussis is an occasional cause of sudden infant death. **Adenoviral infections** are usually distinguishable by associated features, such as fever, sore throat, and conjunctivitis. **Mycoplasma** causes protracted episodic coughing, but patients usually have a history of fever, headache, and systemic symptoms at the onset of disease as well as more continuous cough and frequent finding of rales on auscultation of the chest. Unless an infant with pertussis has secondary pneumonia, the findings on examination between paroxysms are entirely normal, including respiratory rate.

Laboratory finging;

Leukocytosis (15,000–100,000 cells/mm^3) due to absolute lymphocytosis is characteristic in the catarrhal stage. The **chest x-ray** is only mildly abnormal in the majority of hospitalized infants, showing perihilar infiltrate or edema (sometimes with a butterfly appearance) and variable atelectasis. Parenchymal consolidation suggests secondary bacterial infection. Pneumothorax, pneumomediastinum. **Isolation of B. pertussis in culture remains the gold sstandard for diagnosis**. Direct fluorescent antibody (**DFA**) testing of potential isolates using specific antibody for B. pertussis and B. parapertussis maximizes recovery. **Polymerase chain reaction (PCR)** to test nasopharyngeal wash specimens has a sensitivity similar to that of culture, , but is not standardized or available universally.**Serologic tests for detection of antibodies to B. pertussis antigens** in acute and convalescent samples are the most sensitive tests in immunized individuals and are useful epidemiologically.

Treatment;

Infants <3 mo of age are admitted to hospital almost without exception, as are those between 3–6 mo unless witnessed paroxysms are not severe, and those of any age if significant complications occur. Prematurely born young infants and children with underlying cardiac, pulmonary, muscular, or neurologic disorders have a high risk for severe disease. The specific, limited goals of hospitalization are to (1) assess progression of disease and likelihood of life-threatening events at peak of disease, (2) prevent or treat complications, and (3) educate parents in the natural history of the disease and in care that will be given at home. Heart rate, respiratory rate, and pulse oximetry are monitored

continuously with alarm settings so that paroxysms can be witnessed and recorded by health care personnel. Detailed cough records and documentation of feeding, vomiting, and weight change provide data to assess severity.

Typical paroxysms that are not life threatening have the following features: duration <45 sec; red but not blue color change; tachycardia, bradycardia (not <60 beats/min in infants), or oxygen desaturation that spontaneously resolves at the end of the paroxysm; Infants whose paroxysms repeatedly lead to life-threatening events despite passive delivery of oxygen or whose fatigue leads to hypercarbia require intubation, paralysis, and ventilation.

Mist by tent can be useful in some infants with thick, tenacious secretions and excessively irritable airways.

The risk for precipitating cough by nipple feeding does not warrant nasogastric, nasojejunal, or parenteral alimentation in most infants.

Antibiotics.

An antimicrobial agent is always given when pertussis is suspected or confirmed primarily to limit the spread of infection and secondarily for possible clinical benefit. Macrolides are preferred agents. Azithromycin is the preferred agent for use in neonates. All infants <1 mo of age treated with any macrolide should be monitored for symptoms of pyloric stenosis.

Recommended Antimicrobial Treatment and Postexposure Prophylaxis for Pertussis, by Age Group as in the table;

	PRIMARY AGENTS			**ALTERNATE AGENT**
AGE GROUP	**Azithromycin**	**Erythromycin**	**Clarithromycin**	**TMP-SMZ**
<1 mo	Recommended agent. 10 mg/kg/day in a single dose for 5 days (only limited safety data available)	Not preferred. Erythromycin is associated with infantile hypertrophic pyloric stenosis.	Not recommended (safety data unavailable)	Contraindicated for infants aged <2 mo (risk for kernicterus)
1–5 mo	10 mg/kg/day in a single dose for 5 days	40–50 mg/kg/day in 4 divided doses for 14 days.	15 mg/kg/day in 2 divided doses for 7 days	Contraindicated at age <2 mo For infants aged ≥2 mo.
Infants (aged ≥6 mo) and children	10 mg/kg in a single dose on day 1 then 5 mg/kg/day (maximum 500 mg) on days 2–5	40–50 mg/kg/day (maximum 2 g/day) in 4 divided doses for 14 days.	15 mg/kg/day in 2 divided doses (maximum 1 g/day) for 7 days	TMP 8 mg/kg/day, SMZ 40 mg/kg/day in 2 divided doses for 14 days

Isolation.

Patients with suspected pertussis are placed in respiratory isolation with use of masks by all health care personnel entering the room. Children and staff with pertussis in child-care facilities or schools should be excluded until macrolide prophylaxis has been taken for 5 days.

Care of Household and Other Close Contacts.

A macrolide agent should be given promptly to all household contacts and other close contacts. The same age-related drugs and doses for prophylaxis are used for treatment. Close contacts <7 yr of age who have received fewer than 4 doses of pertussis-containing vaccines should have vaccination initiated or continued to complete the recommended series. Children <7 yr of age who received a 3rd dose >6 mo before exposure or a 4th dose ≥3 yr before exposure should receive a booster dose. Individuals ≥9 yr should be given a Tdap (adolescent/adult formulation of tetanus and diphtheria toxoids and acellular pertussis) booster if they have not previously received Tdap and >2 yr have passed since receipt of a diphtheria-containing vaccine.

Complications;

Infants <6 mo of age have excessive mortality and morbidity, with infants <2 mo of age having the highest reported rates of pertussis-associated hospitalization (82%), pneumonia (25%), seizures (4%), encephalopathy (1%), and death (1%). Infants <4 mo of age account for 90% of cases of fatal pertussis. Preterm birth and young maternal age are significantly associated with fatal pertussis.

The principal complications of pertussis are apnea, secondary infections (such as otitis media and pneumonia), and physical sequelae of forceful coughing. The need for intensive care and artificial ventilation is usually limited to infants <3 mo of age. Respiratory failure due to apnea or secondary bacterial pneumonia are events precipitating intubation and ventilation. Progressive pulmonary hypertension or hemorrhage (especially in very young infants) and secondary bacterial pneumonia are usual causes of death. Fever, tachypnea or respiratory distress between paroxysms, and absolute neutrophilia are clues to pneumonia. Children who have pertussis before the age of 2 yr may have abnormal pulmonary function into adulthood. Increased intrathoracic and intra-abdominal pressure during coughing can result in conjunctival and scleral hemorrhages, petechiae on the upper body, epistaxis, hemorrhage in the central nervous system (CNS) and retina, pneumothorax and subcutaneous emphysema, and umbilical and inguinal hernias. Laceration of the lingual frenulum is not uncommon.

CNS abnormalities occur at a relatively high frequency and are almost always a result of hypoxemia or hemorrhage associated with coughing or apnea in young infants. Seizures are usually a result of hypoxemia, but hyponatremia from excessive secretion of antidiuretic hormone during pneumonia can occur.

Prevention ; Universal immunization of children with pertussis vaccine, beginning in infancy with periodic reinforcing doses, is central to the control of pertussis.